LECTURES ON DISEASES OF THE NERVOUS SYSTEM.
SECOND SERIES.

SUBJECTIVE SENSATIONS

OF

SIGHT AND SOUND,

ABIOTROPHY,

AND OTHER LECTURES.

BY

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PREFACE.

The following Lectures, which have appeared in print at various times, have been carefully revised. The first lecture has been throughout rewritten, and its substance rearranged, in the light of later experience, which has not, however, modified its chief conclusions. These can only be extended by the collection of many other facts observed with care and recorded with precision. To promote such observations is one object of its republication. The second has also been carefully revised, and will likewise, I hope, promote more systematic study of a symptom which is apt to be passed over as too common to merit attention. Of the other lectures I need only say that the last, "On the Use of Drugs," although given some years ago, seems to need no change in consequence of fresh knowledge. There is indeed a prospect that our ultimate conceptions may be thrown into crucible, and recast by the influence of ardent thought, through the discoveries regarding radio-activity, and the possible nature of the elementary constituents of matter. But we must remember that these do not alter the coarser facts we were previously able to perceive. All that has been demonstrated regarding atoms and molecules, chemical affinities, and chemical compounds and combinations, and the relation of these to energy, latent or released, remains true, whatever may be discerned of the ultimate nature of the processes.

W. R. G.

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AND

OTHER LECTURES.

LECTURE I.

SUBJECTIVE VISUAL SENSATIONS.

BEING

THE BOWMAN LECTURE,

Delivered to the Ophthalmological Society of London, on Friday, June 14th, 1895.

[The substance of this lecture has been rearranged, and it has been rewritten throughout.]

Mr. President and Gentlemen:—The name of Bowman is associated alike with physiology and ophthalmology. The subject I have chosen for this lecture, given in grateful honour of his memory, combines these two subjects with which that memory is linked. Apart from the results he achieved, already dimmed by the mists which so quickly veil the past, his name brings before us a singular grace of character, and strenuous pursuit of the highest standard of scientific work, a standard we may not hope to reach, and yet may do better than otherwise if we keep it clearly in our view.

The subject I ask you to consider to-day is that of the visual sensations that occur without external cause, sensations of subjective nature. They are not rare, yet definite facts regarding their features are few. This is not surprising. That which is subjective can only be ascertained through the consciousness of another. We have to depend on description to enable us to conceive it; very rarely is depiction possible. Each presentation can be given us only by means of memory; the memory of any sensation is imperfect, and especially so when the visual sensation coincides with mental confusion. Hence the opportunities to obtain trustworthy facts are rare, and when secured they should be utilised to the utmost.

In two diseases subjective visual sensations occur with special frequency, migraine and epilepsy, and in each they are often constant and precise. To these I propose to limit what I have to say and suggest, both for present thought and for future observation.

PHYSIOLOGICAL CONSIDERATIONS.

These subjective sensations present one relation which transcends all others in importance. This is the apparent relation of the sensations to the functions of the cerebral centres in which we must assume that they arise, and through which we must also assume conscious vision of external objects is achieved. In 1885 I put forth the opinion, which has been confirmed by all that I have since been able to perceive, that, in addition to the half-vision centre in the occipital lobe, demonstrated by Munk, the indications obtained by Ferrier are correct, and that there exists a higher visual centre in the region of the angular convolution, a centre which immediately subserves the perception of visual impressions. Impulses seem to pass to this higher centre in each hemisphere from both half-vision centres in the occi-

pital lobes, in such a way that in each higher centre both fields of vision are represented, but that of the opposite eye in greater degree. The connexion between the two higher centres is very intimate, and their function differs from that of the lower in two ways. First, it seems to present the mysterious feature that, while partial disease of the lower centre causes partial local loss of the related half-field, which cannot be compensated, partial disease of the higher centre seems to lower the function of the whole, and there is some capacity for compensation by the other hemisphere, although far less than in the lower animals. This may be one reason why the pathological evidence of this centre in man is so scanty, and another cause may be found in the fact that the centre is in the region of the blood-supply of two different arteries, and so is seldom entirely destroyed. In the strange hemianæsthesia of hysteria we have evidence of arrested action of all the higher special sense centres in one hemisphere. In this association we never meet with the hemianopia that results from disease of the lower centre in the posterior part of the hemisphere. Instead, we have the "crossed amblyopia," as it is termed; peripheral vision is lost in both eyes, central vision persists in a small area in the opposite eye, and in a larger area in the eye of the same side. No trace is found of any relation to the half-fields. But hysteria is a by-word, and all facts, however definite, with which the name can be connected, are disregarded by physiologists. Yet they are definite, and must depend on equally definite functional arrangements. Moreover, the same affection of vision occurs in organic disease. I have given an illustration of it,* as a lasting symptom, in a case in which it was caused, with hemiplegia, by a sudden lesion, probably hæmorrhage, in

* Manual of Diseases of the Nervous System, vol. ii, second edition, Fig. 83 (first edition, Fig. 81).

one hemisphere. A similar loss has been met with when the angular region was found diseased.* When hemianopia is caused by disease of the cerebral hemisphere there is sometimes a like contraction of the remaining half-fields, greater in the eye of the opposite side.† These may seem meagre grounds for the assertion that here we have a higher visual centre, but they agree in a remarkable way with the experimental results obtained by Ferrier, and a large number of the facts of disease cannot otherwise be understood. A theory that explains is not necessarily true. But if a theory affords an explanation which can be obtained in no other way, and has other varied and quite independent evidence, it seems to me that we are justified in its assumption. I have to ask you to postulate these higher visual centres, for I can best describe the facts of subjective sensation, and suggest their significance, by placing them in front of this hypothesis.

Note the necessary significance of "crossed amblyopia." With loss of the higher visual centre of the left hemisphere, we have, in the right eye, vision only in a small central region; in the left eye, sight remains in a larger central region. In each eye there is peripheral loss. The significance of this is that the vision which remains is subserved by the right higher centre acting alone. This subserves a large central region on the opposite side, a small central region on the same side, but, without the co-operation of the opposite centre, it cannot subserve peripheral vision in either eye. In the central regions of the fields, vision can be subserved by either hemisphere alone; in the peripheral parts, by neither alone. It is as if the impulses from the periphery of each retina ultimately reached the higher centre of one side through

* *Ibid.*, Fig. 15 (first edition, Fig. 14).
† *Ibid.*, Fig. 82 (first edition, Fig. 80).

that of the opposite side,—as if those from the peripheral region of each half-vision centre in the occipital lobe passed to the opposite hemisphere indirectly, through the higher centre of the same side, and those from the central region passed directly from the half-vision centre to each higher centre. So that, for instance, the peripheral impressions, to reach the right higher centre, have to pass through the higher centre on the left side, and the loss of this involves peripheral blindness on both sides. Whether this is so, or whether some other form of co-operation takes place, the facts show that the functional relation of the hemispheres is extremely close. It will be remembered that through the optic tract pass the fibres from the same side of each retina, receiving impressions from the opposite half of the field of each eye, and also from the whole of the small central region of the macula lutea, subserving central vision around the fixing point. The conduction by each tract is to the occipital region of each hemisphere, the half-vision centre. Each of these lower centres receives the impulses from the whole of the corresponding half of each retina, *i. e.* the impressions from the opposite half of each field, and also from the whole macular region, so that destruction of either causes hemianopia up to the middle line except around the fixing-point, leaving central vision unimpaired. But the intimate connection between the visual centres of the two hemispheres is shown by the fact that an acute lesion in one lower, half-vision centre, which causes enduring hemianopia, may also cause for a few days absolute loss of sight, complete loss in both eyes, which can only be explained by a process of irritative inhibition of the visual centre on the undamaged side. This is of great importance for our present subject as a proof of the close connection of this function on the two sides, even in relation to the lower half-vision centres. Even more intimate must be the functional blending of the two higher visual centres

that we are compelled to assume, although each seems capable of passing into a state of functional inaction.

Colour-Vision.

Subjective visual spectra frequently present colours—red, blue, yellow and sometimes green. One fact regarding these colour-sensations is that they are often referred to the peripheral parts of the fields of vision, and are as distinct there as in the central region, to which their fields are said to be restricted. Green is supposed to have a very small colour-field, having a radius less than half that of the field for white. But, as an illustration of the way in which the subjective sensation transcends altogether the alleged limits of the field, I may mention a case of epilepsy in which the aura of the fit was uniformly a sensation of a green colour over the lower half of the field of vision, from side to side, so that the patient spontaneously said it was "as if he were standing in a field of grass." Such a subjective sensation is incompatible with the small size of the field for green as commonly described. But if an observer stands in an expanse of grass he will perceive the colour almost to the edge of the field of vision, and he will do so even if he covers the central part of the field. It is the same in the case of the blue of the sky; the blue is perceived to the edge of the field. This corresponds with the subjective sensation, but differs from the common statements regarding the colour fields. This difference led me to examine afresh the facts of peripheral colour vision. The results, I need hardly say, are not new, but they are not sufficiently recognised to make it superfluous for me to mention them. They have been known for nearly twenty years, since Chodin * worked at peripheral colour vision in regard

* Archiv. fur Ophthalmologie, Band xxiii; see also Landolt, A Manual of the Examination of the Eyes; and Swanzy, Diseases of the Eye, p. 19.

to saturation. They were described by Landolt, but they do not seem familiar, to judge from the statements commonly made. The field in which red can be seen is described as a central region the limit of which crosses the outer horizontal radius at 45°. This limit is even spoken of as "the boundary of the field for red." But this is only the area in which red can be perceived in an object one centimetre square. The same statement in regard to a still smaller region is made respecting green. But with a larger area of colour it is found that all the colour-fields increase in size and ultimately differ in extent very little from the field for white. On the temporal side red can be seen as such up to 90°.

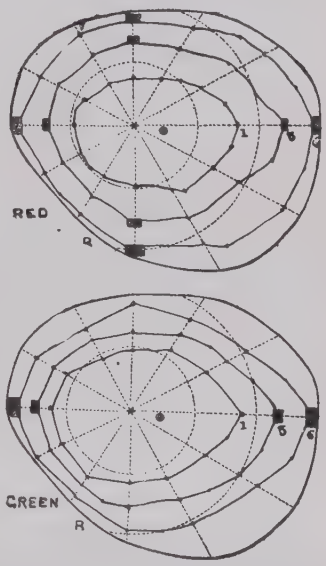


Fig. 1.—The fields for red and green in areas of 1, 5, and 6 centimeters in concentric dimensions, and two-thirds in radial dimension. (The latter is not accurately indicated in the black areas which represent the degrees occupied by the object.)

Let us first take the red field (Fig. 1). It is easy to ascertain that this depends in extent simply on the size of the coloured object. If we take the outer horizontal radius—that is the horizontal radius of the field in the temporal half, the limit of the region in which the colour can be discerned in an area half a centimetre square is, I find, at 35°, and for a quarter-centimetre square at 25° and in one centimetre square at 45°, commonly figured as the limit of the field for red. In an area two centimetres

square, red is seen at 60°, three centimetres square at 70°, four at 78°, five at about 82°, while in an area of six centimetres square the colour is seen up to the extreme edge of the field for white. Upwards, downwards, and on the nasal side of the field, in which the range of vision is so much more limited, the areal limits are closer together, so that on the inner horizontal radius the three-centimetre limit is at 40°, and the six-centimetre area of red can be seen at 50°—that is, at the edge of the field for white. Every field is practically concentric with the field for white. In the case of blue and yellow still smaller areas suffice to permit the colour to be seen up to the edge of the field for white. Green alone seems to fall short of the edge of the white field by about 5°.

Colour.—I have spoken of areas of two or more centimetres square. But this is not accurate. At each distance from the centre at which the colour can be seen in an area of a certain dimension, the same in the radial and in the concentric direction, it can be seen equally well if the radial dimension is only two-thirds of the concentric dimension. For instance, on the outer horizontal radius, red can be seen when an area 3 cm. square has its inner edge on the 70° circle. The colour can be seen equally well if the area three centimetres in the concentric direction is only two centimetres wide, *i. e.* in the radial dimension.

The influence of area is more easy to ascertain than is that of illumination. It is, moreover, of far greater practical importance, on account of the difficulty of securing conditions of illumination sufficiently constant for comparison, and of obtaining colours in sufficient saturation. Of red objects easily obtained, only the petals of flowers will bear reflected sunlight. Pigments reflect so much white light that the colour is often invisible.

I have spoken of the mysterious way in which peripheral vision seems, in the higher centres, to be dependent on mutual

co-operation, that in neither eye can peripheral vision occur unless the centres of both hemispheres are active, while in the central region there is vision in both eyes when only the higher centre in one hemisphere is active. This peculiar mutual influence of the centres is exemplified further by the colour-fields. The field for every colour-area is larger if both eyes are open. This seems natural so far as concerns the part of the two fields which overlap. But it is true also of that temporal part of each field which does not overlap. Binocular intensification extends into this part, and extends into it in the same degree as in the part where the fields are double. For instance, let us take the outer (temporal) horizontal radius of the right field (Fig. 2). In the single field (*i. e.* with the right eye only open, A) red is seen in one centimetre square at 42°, but in the double field (*i. e.* with both eyes open, B) it is seen at 60°, although the left field only extends as far as 56°. In a square of two centimetres, red is seen in the single field at 62°, in the double field at 72°, and in a square of four centimetres it is seen in the single at 80°, while in the double field it can be perceived almost up to the edge at 90°. The fact that in the part of the fields in which each is single (*i. e.* is not covered by the other field) there is increased sensitiveness when both eyes act, gives additional emphasis to the strange fact that, in this region, loss of one higher visual centre causes peripheral blindness. It shows how complete is the blending of function in the two centres. In this outer periphery, vision is impossible unless both higher centres act, but where the rays of coloured light

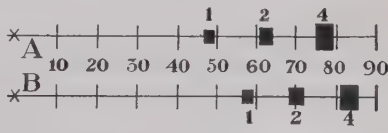


Fig. 2.—The outer horizontal radius of the right field, with the position on it of the limit of the red field for 1, 2, and 4 cm. sq. A, With the left eye closed; B, with the left eye open. The asterisk is at the fixing point and the divisions at 10° distance.

fall on one retina only, their effect is greater if the other eye is open. Further observations show that there is a remarkable extension of each areal colour-field * if a small white area is in the central region, and that this extension is almost the same when it is opposite the other eye only. That is, simple increased stimulation of the central part of either retina augments the sensitiveness of the periphery to colour, and augments it equally whether it is the same or the other eye which is thus stimulated. These facts need more investigation, but they are clearly of importance in connection with the perception of colour at the periphery of the field in subjective sensations. They show that sensitiveness to colour not only involves the centres which are related to the periphery, but can be augmented by the functional activity of other parts. We must recognise the facts, although we cannot understand the process. I have ventured to mention this collateral physiology partly because it is related to the special subject, and partly on account of its intrinsic interest, and its unfamiliarity.

From these physiological considerations, I may pass to the features of morbid action, as they are met with in association with migraine and with epilepsy.

I. VISUAL SENSATIONS IN MIGRAINE.

It is convenient to begin the detailed consideration of the spontaneous visual sensations with those of migraine because they belong to a lower class than do those of epilepsy. They may seem complex in character, but are comparatively simple in nature. The epileptic visual auras are extremely brief, lasting a few seconds, and are followed by loss of conscious-

* The term areal colour-field is convenient as a designation for the field in which colour can be perceived if it occupies a given area.

ness and convulsions. The migrainous sensation is deliberate, occupying a quarter to half an hour, and is followed by a headache lasting for hours. In rare cases similar sensations occur as an isolated symptom, no headache following them.

Not only are the sensations long in duration, but they present peculiar forms. The elementary features are few, but they have special varieties and combinations. They are always "crude" in nature, in the sense that they are low in the scale of sensory perceptions. No visions of objects or faces ever occur. Yet the sensation, simple in character, is remarkable in form, the simple elements develop in the most complex combinations and give rise to spectra that are extremely curious, and will one day, I doubt not, be most instructive. For this instruction, much observation and comparison of facts is necessary. All I can hope to do is to stimulate others to the minute observation through which alone it may be possible to discern the true significance of the phenomena.

Our knowledge of these spectra is chiefly due to the careful description of his own sensations by Dr. Hubert Airy, which appeared in the "Philosophical Transactions of the Royal Society" for 1868. The plates have been reproduced in Dr. Liveing's admirable treatise on Megrim, and an account of the phenomena has been given by Dr. Latham in his book on the disease. I am very fortunate in being able to show you to-night a series of other drawings, unpublished, which Dr. Airy has made of his sensations, accompanied by notes taken at the time. Another series of drawings of great value has been kindly made for me from his own sensations by a distinguished water colour artist, Mr. B——. The peculiarity of the type of sensation and the manifest care of the drawings render them very important. Besides other miscellaneous sources of information I must mention espe-

cially some curious facts recorded by a man named Beck, who when sixty years of age, came under my care as an out-patient at the National Hospital for the Paralysed and Epileptic, and remained so until his death about five years later. During those years he was subject to the frequent appearance of visual spectra, very like those of migraine, but never attended with headache. He was not an educated man, but was a mechanical engineer and mechanical draughtsman. He was possessed by the idea that these spectra were objective things, and he delighted in depicting them in the fashion of an engineering draughtsman. The result was the curious little book which I show you, and hand to the Society for custody.* It contains a number of delineations of these appearances, mechanical in character to an almost absurd degree, yet executed with precision in some features. They are accompanied by quaint descriptions, in which some degree of aphasia can be discerned.

SPECIAL FEATURES OF MIGRAINOUS SPECTRA.

The spectra of migraine present, as their most frequent feature, the zigzag or angled character, which is called the "fortification spectrum" in its well-known curved character. It seems to be due to some opposing forces in the process, by which the discharge in a straight line cannot proceed beyond a short distance and is then compelled to give place to one at right angles to it. But we shall be able to consider its nature better in connection with its features.

The visual discharge of migraine usually presents an association with a process of inhibition of function. The bright appearance is combined with an area in which vision

* This book and Dr. Airy's valuable drawings are in the library of the Ophthalmological Society.

is dimmed or lost, but the relation of the two elements varies and is of great interest. Moreover, the loss of sight may seem to be simple, or may be attended itself by a subdued process of discharge, so that the area in which there is loss may present a dim luminosity, as if occupied by minute particles of luminous sand in constant molecular movement. In some cases the positive discharge is clearly primary and the inhibitory loss is secondary, in others the loss is primary and the luminous discharge is secondary. In the former the bright spectrum usually surrounds the area of dimness of sight; in the latter the primary (or simultaneous) dimness of sight extends to the edge of the field of vision and the discharge occurs within it.

Besides these two distinctions another is constituted by the relation of the symptoms to one side or to both sides of the conjoined fields. The double field, of course, extends equally on each side of the central fixing-point. In one class the leading element is central, and develops around the fixing-point, extending on each side of it. In the other, far more common, the phenomena alike of discharge and arrest of action occur in one half of the field of vision.

It is not easy to conjecture in which visual region these processes occur, whether in the lower half-vision centre or in the higher. The one-sided symptoms seldom pass beyond the middle line, and this fact, together with the simple character of the sensations, may suggest that the process for them occurs in the lower centre. The pain which follows is on the side on which the central function is distributed, that is, on the side of the head opposite to that to which the visual sensations are objectively referred. If these seem to occupy the left half of the field of vision the pain usually begins on the right side of the head. But the symptoms which begin around the fixing-point, and extend equally on each side, can hardly be due to a process in one half-vision centre.

If we assume its seat to be the higher visual centre, it is difficult to understand some of its features except by assuming that both hemispheres are similarly involved. We shall see this better presently. If it seems difficult to conceive an actual identity of functional derangement in the two lower centres, we may remember the fact I have just mentioned, that an irritative lesion in one may cause complete transient inhibition of the other. We may remember also that in all vision there must be perfect combined action of the two. It is certain that the visual impulses first reach these lower centres and that neither receives impressions from beyond the middle line (except just around the fixing-point) and yet there is perfect blending of the two half fields. Although conscious vision must be due to the higher sight centres, the facts seem to show a perfect unity of function in the two sides which lessens the difficulty of conceiving that a similar unity should exist in functional disturbance. At the same time, the correspondence in feature between spectra that are on one side and those that involve both makes it difficult to ascribe some to the lower and others to the higher centres. To this question I shall have to return.

UNILATERAL SPECTRA.

The most common form of pre-migraine spectrum is that which is confined to one half of the visual field. Hence it is convenient to begin with these, especially because we have such a trustworthy record of those forms in the delineations of Dr. Airy.* It is difficult to give exact and convenient designations to any of these forms which these spectra pre-

*I am sorry that the reproductions of these here given are destitute of colour, but this feature is given in the plates accompanying this lecture in the Ophth. Trans. and in Dr. Liveing's book, while many of his original drawings are accessible in the library of the Ophthalmological Society.

sent, but perhaps that which I am about to describe may be termed the expanding spectrum, because expansion is its special feature.

The Expanding Angled Spectrum.

This was the characteristic form experienced by Dr. Airy and the following account of his illustrative drawing corresponds to many others which he has recorded.

A bright stellate object, a small angled sphere, suddenly appears in one side of the combined fields. In Fig. 3 A it is seen a little to the left and below the fixing-point O. It rapidly enlarges, first as a circular zig-zag, but on the inner side, towards the medial line, the regular outline becomes faint (B), and, as the increase in size goes on, the outline here becomes broken (C), the gap becoming larger as the whole increases, and the original circular outline becomes oval. The form assumed is roughly concentric with the edge of the field of vision so far as the lower and outer part of the oval is concerned, where the lines which constitute the outline meet at right angles or larger angles. These remain large and increase as the oval extends, their number and the number of the angles continuing the same. The result bears some resemblance to the place of a fortification and hence it is sometimes called the "fortification spectrum." But (as seen at E) the upper part of the zig-zag oval presents a remarkable difference from the rest. The position of the break is to leave the extremity of this close to the fixing-point of the field (O in all the figures). The expansion above is at first less, so that the upper side is flatter, and the angles lessen progressively towards the fixing-point. Near this they are scarcely to be discerned and at last are represented only by one or two luminous spots. When this angled oval has extended through the greater part of the half field (E) this upper portion also expands; it seems to overcome at last some re-

sistance in the immediate neighbourhood of the fixing-point, although close to it the stability seems too great to be

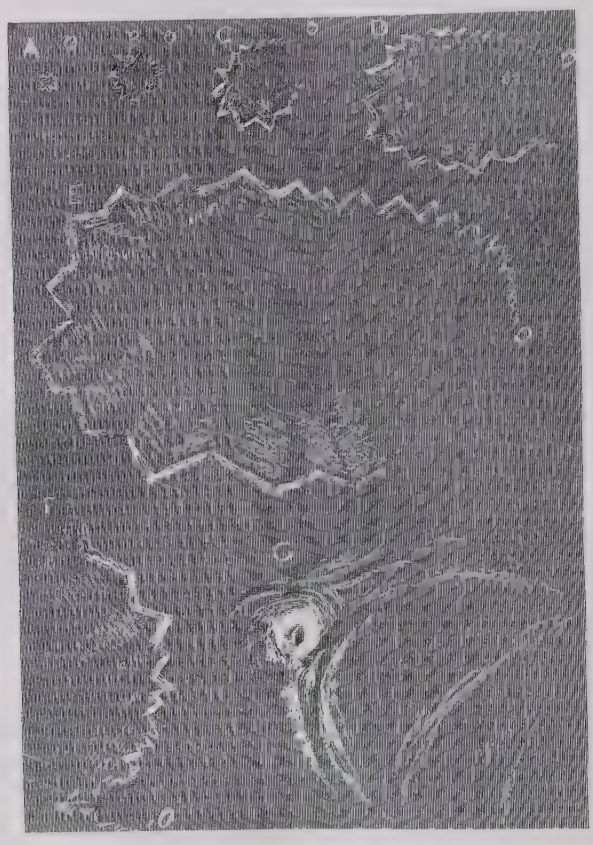


Fig. 3.—The chief elements of one of Dr. Airy's coloured drawings, reduced (Trans. Ophth. Soc.). A B C D E F and G represent the successive stages as described in the text. In all O indicates the fixation point (centre of the field).

overcome, so that a bulge occurs in the part above, and the angular elements of the outline here enlarge (as in F),

although close to the fixing-point the line remains unchanged. After this final stage occurs, the outer lower part of the outline disappears. This final expansion near the centre progresses with great rapidity, and ends in a whirling centre of light from which sprays of light seem flying off. Then all is over, and the headache comes on.

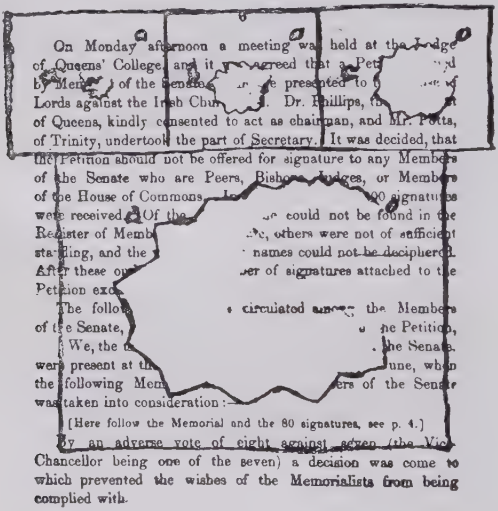
It will be noted that this expanding spectrum does not enlarge to the full extent of the lateral field. Its expansion seems to stop at about the outer third. This is not always the case. In one patient an expanding spectrum developed from a brilliant spot which appeared near the fixing-point, on one side, which enlarged and broke medially so as to form an expanding horseshoe, which enlarged until it apparently disappeared at the extreme edge of the field, for the patient said that it "seemed to pass over the side of the head to the back"—an instance of the apparent objectivity of a peripheral spectrum. In this case the spectrum had not the common angled form, but seemed to consist of intersecting lines, a *cheveux de frise* like that of Mr. B—— presently to be mentioned. Colours—red, blue, yellow—were mingled in the lines, but precise details could not be obtained.

The spectrum of Dr. Airy just described is the most intense manifestation that he experienced. It was often slighter, presenting only a curve, about a semicircle, expanding as the other, but remaining thus limited. This was farther from the fixing-point than in the more intense form, but the upper limb of this also was directed towards the fixing-point and presented the same diminution in the size of the angles, and their ultimate disappearance near the end of the line.

A curious secondary spectrum was sometimes observed towards the end of the process. When the expanding angular outline had attained its maximum, a fresh stellate body was observed near the broken extremities, at or about the

spot at which the first commenced and similar in aspect. Seen for a short time during the fading of the first spectrum, it then disappeared, having the semblance of an abortive attempt to repeat the process.

Colours.—The lines which, in Dr. Airy's experience constituted the angled outline, varied in length, and the luminosity was broken or continuous at their junction; it was


On Monday afternoon a meeting was held at the Lodge of Queens' College, and it was agreed that a Petition ... by Members of the Senate ... be presented to the House of Lords against the Irish Church ... Dr. Phillips, the President of Queens, kindly consented to act as chairman, and Mr. Potts, of Trinity, undertook the part of Secretary. It was decided, that the Petition should not be offered for signature to any Members of the Senate who are Peers, Bishops, Judges, or Members of the House of Commons. ... 900 signatures were received. Of the ... names could not be found in the Register of Members, others were not of sufficient standing, and the ... names could not be deciphered. After these ... number of signatures attached to the Petition ex...

The follow... circulated among the Members of the Senate, ... the Petition,

We, the ... the Senate, were present at the ... June, when the following Mem... bers of the Senate was taken into consideration:—

[Here follow the Memorial and the 80 signatures, see p. 4.]

By an adverse vote of eight against seven (the Vice-Chancellor being one of the seven) a decision was come to which prevented the wishes of the Memorialists from being complied with.

Fig. 4.

often bordered by a narrow dark line. Many of the angled lines presented conspicuous colours, bright red, dark blue, and yellow. The colour occupied the whole of one of the lines or only part of one. The same colour scarcely ever appeared on two adjacent lines. Red was followed usually by blue, sometimes by white, and often a white line had, as it were, a splash of colour in it. It is curious that green was never seen, or at least it cannot have been conspicuous, since it was

never remembered. The only general fact that can be discerned is that the contrast in direction is accompanied by a contrast in colour.

Inhibition.—The loss of sight, in the form of the expanding spectrum, is always within the angled oval. Outside the limiting line, vision is preserved; within it, vision is lost; at first over the whole area; afterwards, when the sphere is broken and has become oval, the loss is most intense close to the limiting line and becomes less towards the middle,

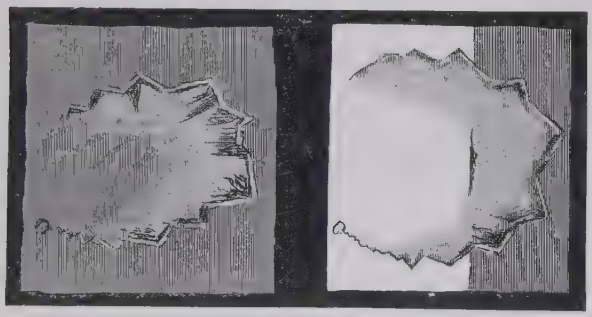


Fig. 5.—Expanding spectrum (Dr. Airy) seen against a dark background, and also half against a light window. The luminosity within the spectrum is seen to be dark, when viewed against the light.

as is well shown in Fig. 4. This represents the observation by Dr. Airy of the print of a newspaper during the development of the spectrum. The luminous zig-zag is represented by the black line.

But the region of inhibition is also one of subdued discharge. When seen in the dark, or with the eyes closed, the region nearest the limiting line, where the interference with sight is greatest, presents a bright scintillation. This is shown in Fig. 5 in which the first figure represents the aspect as seen against a dark background and in the second

figure, the spectrum, a little larger, is seen, half against a dark wall and half against the light of a window. The region in which the light is dimmed is luminous when viewed against a dark background.

More careful scrutiny of this region of faint luminosity shows, as depicted in Fig. 3, that in it there is a peculiar linear appearance; lines of luminosity are ranged parallel to the segments of the limiting spectrum, most distinct near to it. They have somewhat the semblance of internal reflections of the lines, becoming nearer and fainter as they recede from the limiting line, and they are more conspicuous in relation to some of the limiting lines than to others. Unfortunately they seem beyond our present power of interpretation.

THE PROGRESSIVE SPECTRUM.

An angled spectrum may develop progressively through a concentric zone of the half-field, in the position occupied by that just described when fully expanded. If we divide each half-field into three zones, central, middle, and outer, the spectrum has its seat in the outer part of the middle zone. I have only obtained one precise description and delineation of this form. The angular character is marked, but is complex. It develops by progression instead of by expansion, and with this we may connect another noteworthy fact, that the inhibition of function, causing loss of sight, is outside the spectrum and not in it.

In the patient (Mr. B. already mentioned) who has furnished the illustrations, the spectrum was in some attacks in the right half-field, but more often in the left, as depicted in Fig. 6.* The first thing noted (I) was a zone of darkness,

*I regret that the black-and-white reproduction very imperfectly represents the original.

in the periphery of the lower part of the field, diminishing inwards. Then, on the inner side of this, a small star or angled sphere appeared for a minute or two. As it vanished, there developed above it a series of fine angular lines of

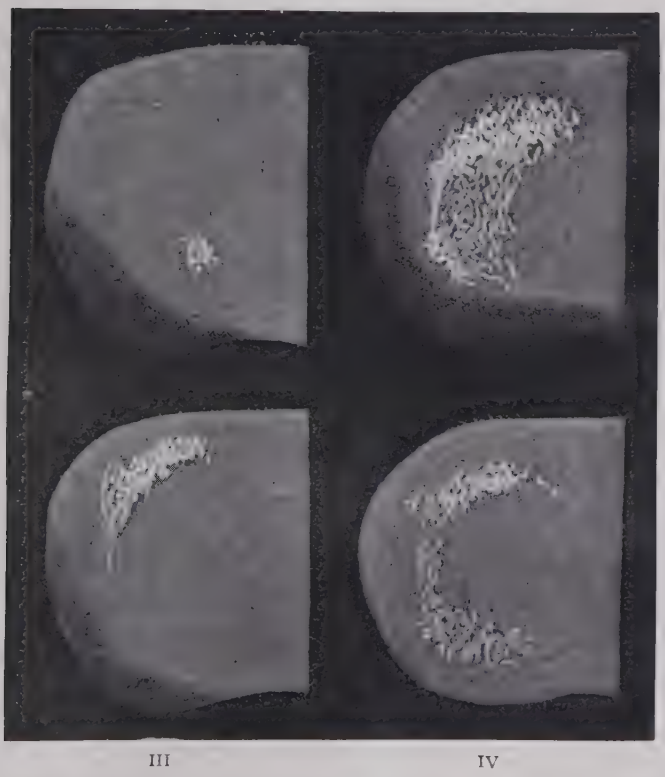


Fig. 6.—Progressive spectrum (Mr. B.). For description, see text.

light, intersecting each other, and progressing in an arc, concentric with the edge of the field, through about a third of a circle. Sometimes the progress of the band of intersecting zig-zags would be interrupted by a flash-like line (II). The

chief development occurred in the upper part of the half-field (although the initial star was in the lower part). Here the intersecting lines constituted the broadest part of the band and in some of the spaces small circles of light appeared. In this part also colours were faintly perceived, red and blue, in the intersecting lines. Within the curve, especially below, much fainter intersecting lines were seen, forming a zone, which had some analogy to the luminous lines within the expanding spectrum described above as presenting the similitude of a reflection. The lower part of the spectrum (first developed) then slowly faded, the upper part remaining bright although narrower. As this began to fade, a fresh development downwards took place, the first indication of which is seen by the downward line in III. This progressed to an extensive development of faint intersecting lines in the lower quadrant, as if by an inverted recrudescence of the primary process, and it was attended by a progressive increase of the peripheral darkness. The brighter region above, however, became smaller in area, and finally all faded away as intense headache began in the opposite temple.

Especially noteworthy in this form are the zonular progression and the fact that the inhibitory loss was peripheral, and began in the neighbourhood of the initial spectrum although not in the part in which the chief spectral process was seated. There is thus an essential difference between these two forms, the expanding and progressive spectra, in the relation to them of the loss of function. Yet the difference is lessened by the remarkable change in the spectrum experienced by Mr. B. The lateral progressive spectrum changed to one of pericentral expanding form, still attended by peripheral inhibition, but with a central subordinate discharge. It will be presently described.

RADIAL SPECTRA.

The two forms just described present one common character—the development of the spectra, in their general course, is concentric; it presents a definite correspondence with the outline of the field. A marked difference is presented by the spectra which have a course from the centre towards the periphery, a course which may be termed radial.

The adjacent figure (Fig. 7) is a representation of a diagrammatic drawing made by a patient. An area of darkness developed in the outer lower part of the field; within this was a bright zig-zag of uncoloured light. The direction of the series of lines is radial towards the fixing-point, the component lines being of course across this direction. It is remarkable that these are smallest and closest together at the periphery, and cease before reaching the edge of the darkness. It is an illustration of discharge within inhibition.

Fig. 7.

In another case a similar radial movement was presented by a stellate object which remained unchanged throughout. It appeared usually near the edge of the right half of the field just below the horizontal line, and consisted of about six pointed leaflike projections, alternately red and blue (another example of contrasted colours in adjacent elements). It appeared on a small area of darkness, as shown in Fig. 8, I. This stellate body moved slowly towards the left and upwards, passing above the fixing-point, to a little beyond the middle line, then it returned to its starting place, retraced

this path once or twice, and then passed to the right edge of the field, keeping in the same direction, so as to come near the edge of the field at the lower outer part. Then it passed back again, only to a little beyond the spot at which it appeared, and then returned to the edge; after two or

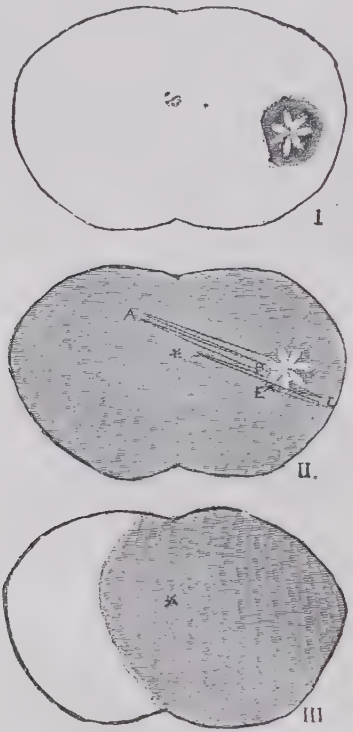


Fig. 8.—Mobile stellate spectrum (see test)

three repetitions of the last course it suddenly disappeared at the spot at which it commenced. The patient kept the eyes shut during its existence, but on opening them when it had gone, always found she could only see in the part of

the field through which the spectrum had not passed. If she looked at a face a couple of feet from her she could only see the person's ear on her left side, and all that was to the right of the ear could not be seen; in only the third of the field to the left was there vision. The loss of sight lasted about a quarter of an hour, and gradually passed away. It is an instructive instance of the relation of the inhibitory loss to the region of the field in which the spectrum appears. The case shows how the central disturbance, though primarily unilateral, may pass beyond the medial line.

This patient, after suffering from migraine for many years, became the subject of epilepsy, the migraine ceasing when the epileptic fits commenced. This transition is not infrequent, and that it was really such is shown by the unusual fact that the visual prodroma of the attacks of migraine became the warning of the epileptic fits, in more rapid evolution.

Hemianopia.—The inhibitory loss of sight, in the case last mentioned, passed beyond the medial line; in the other cases illustrated the loss occupied only part of one side. True hemianopia, complete loss of one half of the field, is often described, but I have never been able to satisfy myself of the occurrence of loss up to the middle line such as results from organic disease. Patients often state that they can only see one half of a face at which they look, but when questioned it is evidently only the greater part of one half that is lost. Partial dimness of one side is all I have been able definitely to ascertain, but the subject needs further careful investigation.

CENTRAL SPECTRA.

The spectra and inhibitory loss hitherto considered are essentially one-sided. They begin and extend in one half

of the double field, and if they pass beyond the medial line it is to a slight degree and in the last stage of development. Indeed, the features of the spectrum of Dr. Airy show the resistance which the central region seems to present to process of discharge. In some cases, however, the disturbance seems essentially central. One example of this was presented by a member of our own profession, whose observations were made with care and may be trusted. At the fixing-point a small round or oval spot of darkness appeared, enlarging rapidly and becoming more intense in the center Fig. 9, A). It extended most rapidly upwards and downwards, and more slowly laterally, until the dark area of loss of sight occupied the whole medial third of the conjoined fields, as simple loss. Its edge on each side presented the form of a double curve (Fig. 9, B). It thus caused blindness, objects being seen only in the unobscured outer side of each field. It often remained thus, without change, until, with the onset of the headache, it passed away. Such a central loss, so perfectly symmetrical, seems inexplicable by an assumed disturbance of the function of one hemisphere. It can only be explained by a simultaneous inhibition of the structures in each hemisphere related to central vision. Just as they must co operate perfectly in normal sight, producing a unity of effect, so we can conceive that they may undergo simultaneous functional inhibition, perfectly symmetrical.

But in this case, on many occasions, the course of the central loss was varied by a remarkable and instructive complication. When the central spot of inhibitory darkness had extended through about the middle third of the vertical dimension of the field (Fig. 10, A), it became limited on one side or the other, but always only on one, by a bright zig-zag line of light. This is seen in Fig. 10, B (where the whole field is dark because the distress compelled closure of the eyes). The line sharply bounded the central blackness on the

left side. It was an angled white line of light, without colour, but with a narrow black edge on each side. As the central cloud increased, the bright limiting zig-zag increased in length,

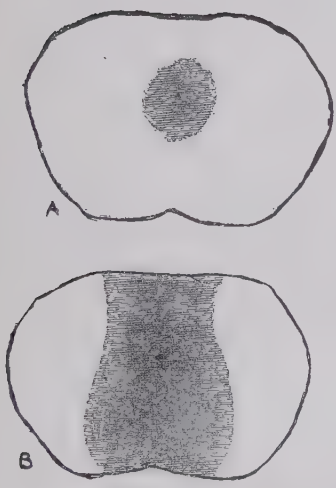


Fig. 9.

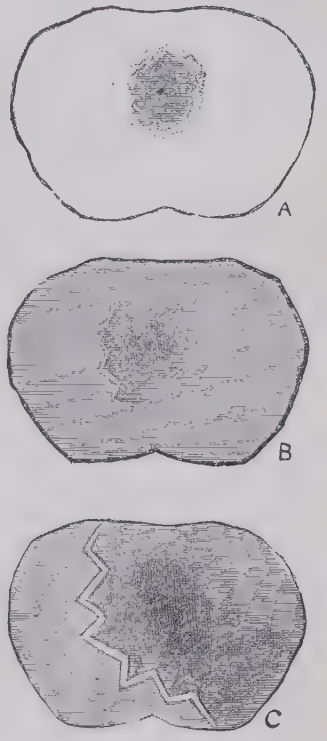


Fig. 10.

Fig. 9.—Medial inhibitory loss of sight, before headache, beginning as a spot around the fixing-point (A), and then extending from the top to the bottom of the double field (B).

Fig. 10.—From the same case. A. Central inhibitory loss around the fixing-point in the double field. On account of the distress caused by it the eyes were closed, but the central blackness was still distinct on the dimmer background, and became limited by a zig-zag line of light (B). This increased in brightness and width, the angles remaining little changed (C). A limiting line of greater darkness was distinct on each side of the line of light, and the inhibitory darkness extended farther towards the opposite side of the double field.

passing below to the other side of the middle line, but it did not move outwards much beyond the place at which it was first perceived. It seemed as if the discharge constituted an obstruction to the further extension of the inhibition, while on the other side the loss of sight spread rapidly, further than it did if there was no bright discharge, and almost reached the extreme edge of the field. The resistance on one side seemed to cause the inhibition to spread farther on the other side, and we must perceive in this the intimate co-operation of the two hemispheres. Whenever the limiting bright spectrum appeared, the headache which followed was always on the side opposite to that to which the spectrum was referred. We may thus assume that the pain was on the side of the hemisphere chiefly in the process of discharge.

PERICENTRAL SPECTRA.

A luminous spectrum seems never to develop at the fixing-point itself. The nearest approach to it is the development of an angled circle around the fixing-point. A coloured zig-zag around the object at which he was looking was occasionally experienced by the man Beck, and his description and drawings of this are so precise as to be evidently trustworthy in general features. When sitting down to dinner, the zig-zag spectrum, coloured red and blue, suddenly appeared, surrounding the edge of the plate before him. The quaint description he gives of it is: "I remember well the phenomena appearing on the plate when I sat down to dinner. As I looked curious my wife said, 'Why do you not carve?' On taking my eyes off the plate I said to them, 'The zig-zag rainbow colours are gone out of the window. This was the first time my wife and friends believed I saw something very extraordinary." One of his drawings shows the coloured angled spectrum surrounding the edge of the plate, and

another the same circular spectrum in a pane of the window at which he looked up. Concentric with the plate as first seen, it maintained the same form when he raised his eyes to the window, and then disappeared. This fact is more important than may at first sight appear, because the form of the spectrum was evidently determined by the actual stimulation of the visual centres.

A change in the essential character of the spectrum is rare, but, in the case of Mr. B. (as already mentioned) the unilateral progressive spectrum, shown in Fig. 6, has undergone transformation with one of the pericentral expanding type, a rough illustration of which is given in Fig. 11. The fact is of great interest and importance. The first symptom of an impending headache is a darkness in the lower part of the double field, which ascends to the central region so as to cause there a cloudy dimness, so that a large object is seen only in parts, which are constantly changing. Then the darkness below lessens, as it simultaneously develops round the entire periphery of the field. At the same time a small bright circle of angled lines appears around the fixing-point, and at the same time the central cloudiness disappears. The angled lines are irregular, but equally so in the whole circle. They present no progressive change as in the expanding spectrum of Dr. Airy (in which the angles of the broken oval lessen in the limb directed towards the fixing-point). This bright angled circle steadily expands until it ultimately occupies more than the middle third of the field, measured from side to side. Its shape being nearly circular, it is nearer the top and bottom than the sides of the oval double field. Between it and the darkness which lines the edge of the field, is a zone in which vision is preserved, which peripherally passes gradually into the bounding dimness, but extends centrally up to the luminous circle. Soon after this is perceived, as it enlarges, the area within it pre-

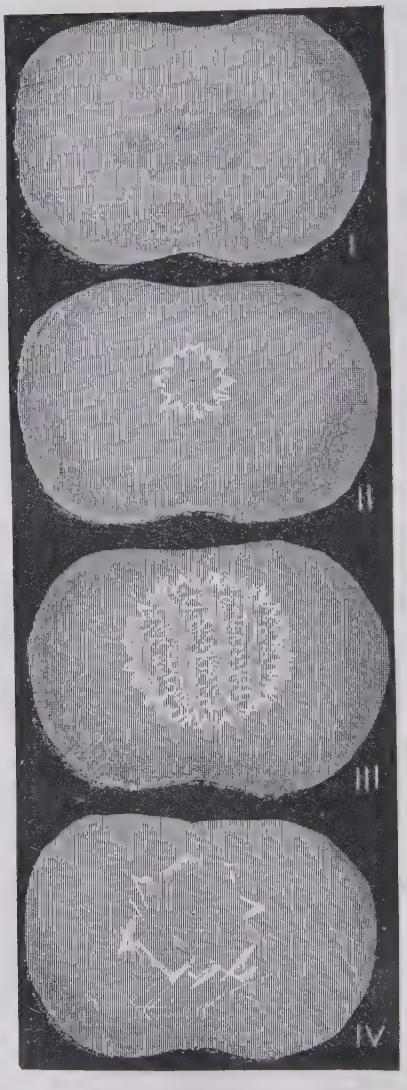


Fig. 11.—Pericentral spectrum of Mr. B—— described in text.

sents a luminosity which constantly changes, but presents a definite character as the circle becomes large. In spite of its mutability, a series of vertical and transverse lines can be discerned, leaving between them small squares, spaces occupied by small circles of light. It must be understood that this is only the impression produced by the changing kaleidoscopic spectrum. The interest of this feature is the greater because similar luminous circles were a feature of the earlier one-sided spectrum, seen in the interspaces of the intersecting *cheveux-de-frise* lines. They were contained by diagonal intersecting lines in the unilateral progressive spectrum, but they are contained by the rectangular lines within the central expanding spectrum.

Ultimately this peculiar central luminosity disappears; the peripheral darkness becomes less, the angles of the circle become larger and larger, and at last are broken, and it disappears. The darkness around the field passes away first at the sides and lasts longest where it began, below. With the disappearance of the spectrum the headache comes on, which, strange to say, is still unilateral.

A spectrum in the form of an arch, in the mid-position, above the centre, was also a frequent experience of Beck, and may be regarded as a segment of a pericentral spectrum. He has given a representation of this, in his usual objective style, of which the adjacent figure (12) is a reproduction. In another drawing it has become divided in the middle, with further loss of some of the constituent colours on each side. It is of great interest that the pericentral spectrum of Mr. B.—— (Fig. 11) has lately been limited to the upper half of that represented, a semicircle but with the same intersecting lines and circles. In these forms also we are compelled to think of the perfect co-action of the two centres.

A form of this arched spectrum was described by another

patient as a sort of angled crown above the eye. It is another instance of the tendency to regard these spectra objectively. The patient was a member of our own profession, and, in response to my request for a drawing of the aspect the spectrum presented to him, he sent me an objective

Fig. 12.—Arched spectrum (with the colours of the rainbow) as represented objectively by Beck.

representation of his eye with the spectrum above it, in the position in which it could, of course, only be seen by another person. This involuntary sense of objectivity is a very

curious feature. It is especially strong when the spectrum is at the periphery, in the part of the field where vision is least distinct. It may be that a vivid sensation, where sensibility is relatively dim, seems so anomalous as to appear absolutely objective.

These descriptions of the leading varieties of the migrainous spectra may at least serve to point to the features to which observation may be usefully directed. The slower development of the symptoms enables much more careful observations and more precise record than in the case of the very rapid and brief spectra which precede epileptic fits, to which we now pass.

II. VISUAL SENSATIONS IN EPILEPSY.

A visual sensation or visual loss is common as the immediate warning of the onset of an epileptic fit—that is, the first effect on consciousness of the cerebral process. Loss of sight, observed as sudden darkness, is always bilateral. In epilepsy there is never simple one-sided loss, not even such partial hemianopia as occurs before migraine. Indeed, the only case I have met with is the case described above, in which the star-like object, moving to and fro obliquely, in an area of dimness, was the premonition of the attacks of migraine and also the warning of the epileptic seizures which afterwards replaced the headaches.

Some positive visual sensation is, however, a common warning. Such sensations are very varied, but they are, as a rule, constant in character in the same patient. They may be quite simple, as simple as those which precede migraine, but they never present the peculiar angled form which is so common before headache. They are, moreover, extremely rapid in evolution, and brief in duration. Just as the epileptic fit occupies fewer minutes than the hours during which

the headache lasts, so the warning sensation occupies fewer seconds than the minutes during which the visual premonition of migraine continues.

The simplest forms are as follow: (1) A sudden appearance of a light or colour, compared sometimes to a flash, sometimes to a slower glow. It is usually general, but occasionally seems to occupy part of the field, upper or lower. Strange to say, it is seldom seen on one side. An example of this has been already mentioned; the uniform warning of every fit was the appearance of green through lower part of the field of vision, from side to side, spontaneously described as if the patient was "standing in a field of grass." (2) Sparks or stars, small and often numerous. If very numerous, they may appear in rapid molecular movement, like insects on a pond, such as already described as a feature of the secondary discharge of migraine. (3) Sometimes a larger luminous object is seen, described as round, not as definitely stellate, often coloured. It may be of a different colour in the centre and margin; in one instance the constant aura was a bright spot, blue in the middle and red around. In some cases the colour is always the same; in one it was brilliant blue and was induced by looking at a bright light of any kind. But the colour may change, as from red to green. Sometimes a simple light may change to some definite objects. A light may move across the field or seem to approach or recede. A common form of movement is circular. A bright light appears and moves round and round, either seeming to come nearer by movement in an enlarging spiral, or to recede; after a few revolutions, consciousness is lost. In one case, in which the revolving light approached, as consciousness began to fail and the light to become dim, a most offensive smell was experienced, always the same, but indescribable. Such rotation seems to be a sensory analogue to the form of vertigo in which seen

objects have a similar circular motion. The fact that a subjective visionary object may seem to move is of much significance and needs special consideration.

SIGHT AND MOVEMENT.

The apparent motion of spectral objects illustrates the close relation of sensory and motor processes. Any sensation on the surface will cause an instant movement of the head and eyes in that direction. So will a sound that is heard on one side: the head is instantly turned in that direction. So an object, seen on one side, induces an instant movement of the head and eyes towards it, so as to bring it to the place of most distinct vision. I remember once seeing an instance of this: a number of wild swans, fifty or sixty, were floating down a river, when a dog appeared on the bank some thirty yards behind them. Instantly every swan was in the same position, each neck presented the same curve, each head had the same inclination, so as to bring one eye of every swan to bear upon the dog. So they remained, in the same position, which slowly changed as the dog passed by. The correspondence of posture and movement was a striking illustration of the uniformity of nervous action under the same visual stimulus. In man, in the same way, an impression on the retina on one side of the field of vision causes an instant movement of the head and eyes towards it, to bring its image to the centre of the field. If the object moves, the head and eyes follow it. This is true of a subjective sensation as well as of one that has a real cause.

There is also an opposite relation between sight and movement. Not only does sight induce motion, but movement of the head or eyes causes an apparent motion of objects that are seen. If the image of a seen object occupies the same place on the retina, while the head and eyes are moved, the

object must have moved in corresponding direction and degree. This is an instant inference from the sense of movement of the head and eyes. But in epilepsy there may be such a sense of movement without actual movement; motor processes are felt which are insufficient to cause motion. From this there results a sense of movement, and a seen object, really still, seems to move in correspondence to the subjective motor sensation. It is so in other forms of vertigo as well as in the epileptic discharge. A sense of turning to the right, without movement, induces the impression that an object opposite the eyes moves to the right, and the individual may be conscious of this impression far more than of the motor process which gives rise to it. Thus the sense of objective movement may be apparently primary when really secondary.

This is most common and most important in the subjective sensation of motion to one side, so frequent in the visual sensations of epilepsy. It is related to the deviation of the head and eyes which is a common feature of the attack, and so often begins it. It must be ascribed to the fact that the discharge is greater in one hemisphere of the brain than in the other, and when it is the earliest motor symptom, it shows that the discharge on one side develops before that on the other. Such turning of the head and eyes to one side is not unfrequently associated with a visual sensation.

If an object actually in motion appears on one side of the field, say on the left, the effect of the sensory impression on the brain is primarily on the right hemisphere. The visual process arouses the motor centres on that side which turn the head and eyes towards the left to "fix" the object. But the object is moving from left to right, and, therefore, to keep the object fixed, the first movement towards the left is followed by one in the opposite direction, due to the left hemisphere, to follow the object, until it disappears on the

right. This long movement to the right is then followed by a return movement to the mid-position, by the action of the right centre, when the exciting object has disappeared.

Note this normal alternation under the influence of stimulation of the visual centre of one hemisphere from the opposite side—motor action first in that hemisphere, then in the other, and lastly in that which was first excited. Such alternation of motor activity occurs in disease as well as in health; it may be associated with a primary discharge in the visual centres, causing a visual epileptic aura.

It may be a useful illustration of what I have said if I mention a case in which the same motor sequence occurred as a result of an organic lesion, traumatic meningeal hæmorrhage over the left hemisphere. It caused a long series of right-sided convulsions with strong deviation of the head and eyes to the right. But this was preceded by their deviation to the left, and before the turning to the left there was initial briefer deviation to the right. The slow-commencing discharge in the left hemisphere, causing this first movement to the right, seemed to excite a stronger activity in the right hemisphere, with deviation to the left, before the final intense discharge of the convulsion with strong deviation to the right. The meningeal hæmorrhage was over the left hemisphere only, and the phenomena of the fits, which I watched for hours, show the very close connection between the motor centres in the two hemispheres, and their disposition to alternate energy, such as they manifest in response to visual excitation.

These facts illustrate the features of a case which is specially instructive. A boy, at the age of five, had a severe general illness, of unknown nature, during which sudden hemiplegia came on. Partial recovery was followed by recurring convulsions of the left, hemiplegic side. Each fit began thus: a slight movement of the head to the left, a

much stronger and longer movement of the head towards the right, then a deviation again to the left, intense, as the boy passed into the convulsions, chiefly on that side. Here we have again the same sequence, and here we have an opportunity of discerning more of the process. The patient came under observation as a most intelligent boy of eleven years of age, and described the following warning: The figure of a man suddenly made its appearance at the extreme left of the field, slowly moving towards the right. He felt compelled to try to look at it and to follow it, and did so until it disappeared at the right edge of the field. Then he lost consciousness, and knew nothing of the subsequent strong deviation to the left in the fit. The movement to the right (to follow the object) chiefly impressed his consciousness, and was felt as conscious turning to the right. Here, then, we have these sequences distinctly associated with the action of the visual centre, and, apparently, with the influence of this on the motor centre. Yet one other case. In this, each fit was preceded by the sudden appearance of a blue light on the left side, sometimes of two or three, near together. These always moved slowly towards the right and a little upwards, and the patient felt compelled to follow them. The head and eyes were moved towards the right in this effort, as far as they could be, by strong tonic spasm in the muscles of the neck. This occupied about thirty seconds, and then suddenly ceased on the disappearance of the visual spectrum. The eyes and head, thus released, could be moved from the right, but this movement was continued towards the left as an involuntary movement, which was, however, far greater in the eyes than in the head. For nearly a minute the eyes were turned extremely towards the left, and the head only half-way from the mid-position. During this time he could move the head towards the right, but was absolutely unable to move the eyes from their extreme deviation to the left.

Then he lost consciousness and the general convulsion of the fit came on. We have here again the same sequence, but the convulsion was not left-sided, it was general, and the final left-sided deviation of the head had not the intensity which accompanies a left-sided fit. I could give other similar instances, but these may suffice to indicate the close relation of the movement of the head and eyes to activity of the visual centres. I may point out how indispensable, for understanding the facts, is the theory of the representation of both fields in the higher visual centres and of their mutual action.

An apparent motion of spectral objects upwards or downwards as part of the visual aura of epilepsy seems to be extremely rare. I have not met with it. An apparent rotation of objects actually seen is often described as a feature of initial vertigo, but this is not within our present subject. Movement in circles has been already mentioned.

Micropsy and Macropsy.

An apparent movement towards or from the subject is not uncommon in the case of spectra, and even of objects that are actually seen. I have already mentioned this fact. Recession is usually associated with diminished size and sometimes with dimness of sight, but of the patients I have questioned, a few have been sure that the apparent recession of seen objects was not the result of their dimness. It may be due to a central condition related to diminished convergence and accommodation. But such a recession is rarely described in the case of subjective sensations. With these, apparent approximation is the rule. A spherical light, for instance, seems to get nearer and larger until it overwhelms the patient as consciousness vanishes. It is noteworthy that such approximation is described only in the case of simple visual sensations, not in those of elaborate character.

Psycho-visual Sensations.

More elaborate sensations often constitute the warning of an epileptic attack, but are unknown in association with migraine. They are definite sensorial conceptions of figures of persons, or faces or scenes. A simple luminous object may change to such, as already mentioned. These are so much higher in the scale of mental processes than the simpler sensations already described that they may be termed "psycho-visual." I have given many instances in my book on "Epilepsy."* It is remarkable that the visual conception, however elaborate, is generally constant, preceding every attack. It may be such as cannot have been within preceding experience. In one patient, for instance, every attack was preceded by a sudden vision of London in ruins, herself the sole survivor in the scene of desolation.

Associations.

Visual sensations may be associated with subjective sensations of hearing, very seldom of smell. The two are often of the same degree of elaboration. If the vision is of a person, words may seem to be heard, although they can seldom be recalled. One strangely complex aura, which preceded every fit the patient had, deserves mention. It began in a simple form. First the beating of the heart was felt, and this ascended the chest to the head, where it seemed to become audible as a sound; then two lights appeared before the eyes and seemed to approach by jerks, synchronous with the pulsation. The lights then disappeared, and were replaced by the figure of an old woman in a red cloak, who offered something that had the smell of Tonquin beans; then

* "Epilepsy," etc., second edition; London, Churchill's; Philadelphia, Blakiston's; 1901.

consciousness was lost. The case is remarkable on account of the change of the sensation of simple pulsation to that of sound,* the association of the latter with a pulsating visual spectrum, the replacement of these by a highly complex vision, and the final termination with an olfactory sensation, probably elaborate. The jerky movement, definitely synchronous with the pulse, is not uncommon in such simple visual sensations, and seems to indicate that the process, in the disturbed centre of the brain, is modified by the mechanical influence of the arterial pulsations. I may point out also the interesting fact that the more elaborate sensation, the vision of the woman, *followed* the more simple one, the two lights. This sequence is more common than the opposite, although now and then an elaborate sensation is followed by one that is more simple.

I have mentioned a case in which a light, approximating by circular movement, was associated with an offensive smell. I have met with other instances in which an unpleasant olfactory sensation was associated with a simple visual sensation, and an agreeable smell with one of complex character.

I have already mentioned the remarkable fact that the sudden spontaneous activity, which we call "discharge" of the centre, is often preceded by sudden arrest of function, by inhibition. It is one of many instances of the fact that the same functional derangement, in different degree, seems to cause cessation of action or increased action. In one case, suddenly all became dark, then there appeared a red light before the eyes, changing presently to green, and then consciousness was lost and the convulsion came on. In another, sight became dim and misty, then two round green lights appeared, compared to a penny at a distance of two feet; these moved from side to side. It is very common for the

* See the following lecture, on "Subjective Sensations of Sound."

first darkness to be the background for bright stars. In these cases there is discharge in a centre first inhibited. Much less frequently, stars or flashes of light are followed by loss of sight and darkness,—that is, discharge is followed by inhibition.

III. MISCELLANEOUS SENSATIONS.

Spontaneous activity of the visual centres, giving rise to a sudden light, or colour, or to the impression of some seen object, sometimes occurs in abnormal cerebral states apart from epilepsy or migraine. But such visual sensations are too rare and too vague in their associations to merit detailed description. As an instance of the anomalous visual impressions that may occur, may be mentioned the case of a woman, aged thirty-seven, who frequently experienced a peculiar colour hemiopia; if one eye was closed and a light was looked at (as a candle-flame) the temporal half appeared blue, the nasal half normal. Some time afterwards only a bluish-purple halo appeared all round the flame. After reading for a few minutes the page of print appeared a light green, and then changed to lilac or pink, and then these colours became mixed and mottled over the page. On resting the eyes, sight became normal, but a few minutes after resuming reading the same colour phenomena occurred. No abnormal condition of the eyes or field could be found.

Vertical Hemianopia.

The blending of the visual centres in the two hemispheres is strikingly evident when subjective functional disturbance has a distribution, not from one side, but in the vertical direction. I have mentioned one instance of this, in which the aura of epileptic fits was the sensation of a green colour throughout the lower half of the visual field. The impression was spontaneously compared to "standing in a field of grass."

In very rare cases the lower part of the fields is lost before migraine, the division being either transverse or oblique. In one case it was preceded by numbness in the upper lip. Coloured zig-zags were sometimes seen, but not on the occasions of the transverse loss. In the oblique passage of the stellate spectrum just described (Fig. 8) we have evidence of a functional disturbance involving both sides of the double fields, which makes it easier to realise that the disturbance of the centres may have such a form as to cause inhibition of the upper or lower regions. In the cases I have met with the loss has been in the lower half.

CONCLUSIONS.

Far more observations, precise and detailed, are needed to enable any definite inference to be drawn regarding the indications of the phenomena that have been considered. Indeed, it is probable that, for a long time, the chief result of more observation will be to point the line in which still more is needed, if real knowledge is to be secured. Frequent as is migraine, and frequent as are the associated visual spectra, the ability to observe carefully such quickly changing phenomena is rare, and their combination with mental distress constitutes a further hindrance. Still more rare is the ability to depict them, and even to describe them with needful precision and fulness. But the example afforded by Dr. Airy, who has added so much to our knowledge, should not be lost on the many members of our own profession who are similar sufferers. We may learn much regarding the action of the cerebral centres from such observations. The features of the spectrum in character and colour, its progress, its initial form, position and development, the way and place at which it disappears, are each and all important. So also are the precise position and relation of the colours that appear.

Not less important is the relation of the positive spectrum to the inhibitory darkness.

Yet the question presents itself again and again, to what part of the visual centres are these phenomena to be referred? The problem has been already referred to, but we may note that one conclusion seems compelled by a consideration of the phenomena—they must, in most cases, depend on a functional fusion of the centres in the two hemispheres. This is obviously the case if they can be regarded as due to a disturbance in the lower half vision centres, and it is equally necessary if we assume that the higher visual centres are the seat of the disturbance—the assumption on which they are best explained.

We have seen that the indication of loss of function is that each higher centre is able by itself to subserve vision in a large area around the fixing-point in the opposite eye, having a diameter of about a third of the field, and of a much smaller area in the eye of the same side, having a diameter of about one-eighth of that of the field. In the rest of the fields vision depends on the combined action of both visual centres. The two hemispheres, acting together, subserve vision up to the periphery of the field. (How real is the mutual co-operation of the two hemispheres is shown by the facts regarding the augmentation of colour-vision mentioned on page 14.) The inference from these facts is that the intensity of functional action in the combined centres lessens from the centre to the periphery. If we divide the double field into three zones, each limited at a third of the radius: we have a central zone around the fixing-point (subserved by the opposite hemisphere acting alone), a larger middle zone, and an outer peripheral zone. The chief development of the expanding spectra seems to occur in the middle zone. This division may at least serve to guide future observations and make them more precise.

The difference between the two forms of expanding spectrum, the one-sided oval of Dr. Airy and the pericentral later form of Mr. B., suggest that in the former the discharge is in the higher centre of one hemisphere, while in the latter it occurs in both, combined by a perfect functional fusion. In the former the first stellate object appeared on one side of the fixing-point, near to it; in the latter, the initial small-angled sphere was around the fixing-point. The area immediately adjacent to the fixing-point seems not to be involved in the discharge. The centre for this, the area of acute vision, in either hemisphere, seems to possess an invincible resistance to primary discharge; although it may undergo inhibition and secondary subactivity.

On the other hand, the peripheral part of the fields, which seem to need the co-operation of both hemispheres for vision, may undergo arrest of function in association with the zonular discharge. It seems to be an associated event, and not a consequence, since it may come first, and is most intense at the edge of the field, lessening towards the spectral zone.

Inhibition within the area of discharge is met with only in the expanding form, and seems a residual derangement of the structures through which the discharge has passed, by which they are the seat of subordinate activity with an inability to receive the impulses that reach them from without. It is a derangement which, intense when the discharge has just passed (causing the semblance of reflection presented in Dr. Airy's drawings, Fig. 3), lessens gradually. In the pericentral spectrum of Mr. B. (Fig. 6) the features of the subordinate discharge within it are remarkable; their uniformity in the enclosed area may be connected with the fact that it is the central region of the field. But the most remarkable relation of discharge to inhibition is the limitation of medial darkness by the spectral barrier shown in

Fig. 10. It is a striking illustration of secondary discharge. The inhibition extended from top to bottom of the double fields, perfectly symmetrical, and can only be conceived as an arrest of action of the combined centres in the two hemispheres, practically one. Often it remained symmetrical medial darkness, until it passed away. Sometimes on one side, presumably in one hemisphere, when the inhibition had almost reached its limit, the process suddenly changed to discharge, a bright zig-zag, like a lightning-flash at the edge of a thunder-cloud. The discharge continued fixed, as an absolute limitation to the arrest on that side, but it spread still further on the other. It seems as if the process that was going on uniformly in the conjoined centres of the two hemispheres suddenly gave place to discharge in one. It is remarkable that the discharge was limited to the side on which the later pain was felt.

The examples of spectra which proceed in what I have termed a radial course, from the edge of the field towards the middle, are too few to permit any inference from their remarkable features. These present a contrast to the spectra of concentric form. They occupy the peripheral region, into which the latter do not pass. But it is noteworthy that the discharge seems to be always within a region of inhibition, and that movement is in opposed directions, so as to produce either a series of close-angled lines, or the aspect of an object moving backwards and forwards. Our need is especially great for more observations of spectra of this class.

The close relation between inhibition and discharge is conspicuous in most of these phenomena. The process of arrest seems to be a slighter degree of that which causes excessive activity. This is conceivable even though they are exactly opposite in nature. If we consider the processes in the light of the neuronic theory, and the dendritic endings as the source of the nerve impulses, we can conceive that a

slight degree of energising may induce their retraction, with inaction as a result; a greater degree of functional activity may cause the impulses to burst through the intermediate substance and induce the approximation of the dendrites in intense action. Certain it is that the relation between arrest and activity is common in all such spontaneous processes. In a slight epileptic fit, due to local disease, the arm may fall powerless, while in a more severe attack it is violently convulsed. So, too, also in epilepsy, sudden darkness may be followed by bright lights. The mobile star or zig-zag just mentioned, within the area of darkness, illustrates the same phenomena.

These processes, as discerned and presented in the field of vision, have to be described in topographical language. We know that such description is accurate as regards the lower visual centres. We know that destruction of a definite part of this centre causes loss of a definite part of the half-field it subserves. But we are not justified in assuming any topographical correspondence of the higher visual centre to the visual field. On the contrary, present evidence suggests that partial damage lowers the action of the whole, and that function is subserved in it in a way to which we have as yet no clue. If so, we must obtain far more knowledge before we can hope to understand the precise features of the spectra described, such as the contrasted direction of the lines and their contrasted colours, which compose the spectrum, whether the lines are continuous or intersecting,—the precise position of the spectrum in relation to the double field, and especially its relation to the fixing-point in its origin and course. Not less important is the position and development of the area of darkness. In the hope that more facts may be forthcoming, I will conclude with a simple diagram of the double visual fields, which can be easily copied or enlarged, on which can be depicted the phenomena observed. The form of the discharge can be indicated by pencil lines,

the area of inhibitory darkness by shading. If a wash of colour is put over it, the bright spectrum can be indicated by scratching off the colour with the point of a knife, and the darkness by shading. An indication of the colours seen, and their position, would add much to the value of the depiction. I hope that from the many who suffer and have the ability to supply the record, some facts may be furnished which will enable us better to understand that which is now mysterious. We cannot tell what additions to knowledge may result from facts that seem simply curious.

The information regarding the cerebral visual functions furnished by organic disease is chiefly negative; it is almost limited to their loss. Only in such functional disturbance as we have considered have we evidence of active functional disturbance. Deranged though it is, we must learn from it much that can be discerned in no other way, and may learn more than we may anticipate. The phenomena seem mysterious, but we cannot doubt that the mystery will lessen as observations accumulate, careful and precise.

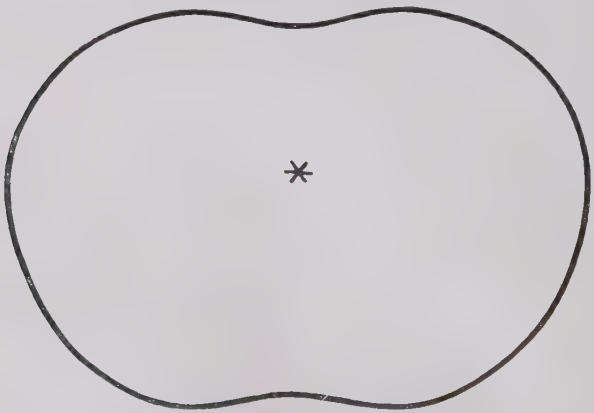


Fig. 13.—Outline of the double field, to be enlarged for the depiction of spectra. The star is in the position of the fixing-point. If this is replaced by a small hole, looked through while the diagram is held near the eye, the relation of the spectrum to this can be best discerned.

LECTURE II.

SUBJECTIVE SENSATIONS OF SOUND.

THE BRADSHAW LECTURE,

Delivered before the Royal College of Physicians of London, November 5, 1896.

I had an opportunity, not long ago, of considering the sensations of sight which occur apart from external cause,—subjective visual sensations. I propose to take the present occasion, afforded me by the honour of the delivery of the Bradshaw Lecture, to consider the analogous sensations of sound,—subjective auditory sensations. Between the two we may trace some points of resemblance and many of difference; but the sensations of hearing merit, not less than those of sight, a careful study. This indeed they have many times received in their relation to the ear, but their general features, especially in relation to the brain, have had less notice. The relations of these symptoms to the sensory organ itself are far more obtrusive in the case of the ear than the eye. Ocular disease rarely causes symptoms of this character, whereas they are among the most common and most distressing effects of disease in the ear. This has been recognised by terminology, popular and medical. "Noises in the Ears" and "Noises in the Head"; "Tinnitus aurium" and "Tinnitus Capitis"—these terms express the prominence to which the symptoms attain.

We speak of these sensations as "subjective" because

they have no external cause,—no cause such as normally gives rise to a sensation of sound. But many of them have a definite cause, external to the structures in which the auditory nerve ends, although not external to the ear itself. Of those that have not such causation, many are due to processes in the structures of the labyrinth in which the auditory nerve ends, and through which the waves of sound excite nerve impulses. Others again arise by a morbid functional action of the centres in the brain through which the impulses from the auditory nerve affect consciousness, and these are subjective in the purest sense of the word, while a few are perhaps due to disturbance at some point in the intermediate conducting path. The subject is thus highly complex. I think I may best present to you its chief outlines if I confine myself to the facts regarding the several forms that have come under my own notice. This limitation does not imply any assumption of originality on my part. I doubt not that many things I have to say have been said before, and to those by whom they have been said I owe this explanation; but it is curious how few attempts have been made to consider these phenomena systematically.

Among the facts that I shall have to mention are some relating to the power of hearing certain musical notes. These are usually indicated by a number for the octave attached to the letter of the note. They may also be indicated by the number of vibrations a second.* For the production of the notes, tuning forks are available up to C^3, and above this we have that most useful aid to clinical observation, a Galton's whistle. By this, high notes can be produced with precision, ranging from 4,000 vibrations (two octaves above the treble clef) to the utmost capacity of the human ear, 20,000

* This is considered, and the most convenient system suggested, in a note appended to this lecture.

or 25,000 vibrations a second, and, indeed, far higher, when a cat will start though there is absolute silence to us. Age lessens the power of hearing these high notes, but the significance of a deficiency that is either early or one-sided is definite, and renders the service of this instrument in diagnosis very great.

Visual and Auditory Sensations.

The resemblance which exists between the subjective sensations of vision and those of sound is superficial; the difference is profound, and it is also instructive. Subjective visual sensations are chiefly of central origin, and in themselves are chiefly of physiological interest, while those of hearing are to a large extent produced in the sensory organ itself and are often evidence of its disease. They are often so obtrusive, and even distressing, that by the sinister dignity of the special name, tinnitus aurium, the symptom has almost come to be looked upon as an independent disease. A definite designation is, however, at least a testimony to the frequency and reality of the suffering that is caused. It would be harmless were it not that names, once called into existence as our servants, are never long content with servitude. Too often we find ourselves controlled by them, and that which is only a symptom, when it has been named, is often looked on as an actual malady.

Sight and Hearing.

The frequency with which the ear, compared with the eye, is a cause of subjective sensations, is also of indirect significance. We are apt to place these two senses too near together in our thought. Both are indeed excited by energy in the form of wave motion. Hence they present

points of resemblance which may attract more attention than does the difference, or rather the contrast, between them. Consider,—for the question is of importance for us,—consider the difference involved in the frequency of the vibrations that act on each organ. If we take a colour about the middle of the visible spectrum and a musical note below the middle of the treble clef, we shall find that, in the interval of time between two successive waves of sound, there occur about one million million waves of light. The statement seems incredible, but it is true, although, like many other facts, it transcends our power of imaginative conception. So does the difference in rate of propagation. Light is transmitted about one million times faster than is sound in air. It is not surprising to find, therefore, that whereas the waves of sound occur only in media so definitely material that they can be weighed, the waves of light occur in a medium which is not only imponderable but almost hypothetical. Further, the waves of light can be traced upwards until they vanish to our direct perception, though beyond them, and their actinic successors, there seem to be waves of such a nature that they have been designated by the algebraic symbol for that which is unknown—the letter x. On the other hand, they pass downwards to the slower waves of heat, which we cannot see and yet can feel. The waves of sound pass up beyond the range of human hearing, though they may still be audible to animals. How far higher they extend we do not know. But the lower notes of music consist of vibrations so slow as to be perceived by the sense of touch if they occur in a medium sufficiently inelastic, and at last they can be only felt, ceasing to be audible except as a series of sounds not musical.

With the enormous difference in time between the waves of sound and those of light, and the vast difference in the character of the media in which they occur, is associated another difference. The waves of light, like the common

waves of water, are transverse to the line of propagation ("transversal" waves, according to the double adjectival form which physicists have revived). But the waves of sound are "direct"; they occur in the line of propagation, as the motion between billiard balls when one at the end of a row is struck and that at the other end moves off. These "direct," or "end-on," waves seem to possess a greater impetus, and such wave motion has a relation to the simple motion of a mass; it resembles simple "contact," by which the nerves of touch are commonly affected. These facts, which point to an essential identity between the stimulus of touch and that of sound, are not disturbed in their significance by the special character of many auditory sensations, such as musical notes. We may note, moreover, the facts of development that show a common (epiblastic) origin for the structures that subserve each form of sensation, hearing and touch, and the facts of anatomy, which show that sound is received by minute hairs that act upon the nerves. We may also note the facts of comparative anatomy, which show the early form of the ear to consist simply of hairs upon the skin connected with a nerve which is homologous with that of hearing in higher animals. We shall presently see in other ways the importance, for our subject, of this relation of hearing to the sense of touch, but I may point out that it removes one apparent anomaly—the fact that one part of the auditory nerve seems to have nothing to do with hearing. Although we cannot say that sound does not act upon the structures of the semicircular canals, we cannot doubt that their chief function is to cause nerve impulses to be produced by the simple motion of the fluid within them, and its varying pressure on the hairs in their dilated ends. We have in this a response to the very simplest form of motion, quite as simple as that which excites the nerves of touch. And I would ask you especially to note the fact that, in the same organ, struc-

tures which closely resemble each other give rise to sensory impulses which seem to us quite different, but may prove to be nearly related. The impulses produced in the semicircular canals do not act upon consciousness, although their effects may do so.

Varieties of Tinnitus.

I may safely assume that the chief features and varieties of tinnitus are familiar to you. You know that tinnitus may be a continuous sound, or pulsating, and that in the latter case the pulsations correspond to those of the arteries. You know also how many and extensive are the variations in its character, from the simplest to the most elaborate sound, from a rushing or roaring sound, or whistling, to that of bells, or music. The frequent coincidence of these sounds with evidence of ear disease, varying in seat and character, but generally involving the labyrinth and often limited to it, compel the conclusion that the most common source of these sounds is the internal ear, in which the auditory nerve is normally excited. But we have cases, especially those in which the sensations are the warning of epileptic fits, in which we must regard the auditory centre in the cortex of the brain as their source, and in rare cases the correctness of this conclusion has been proved by the discovery of organic disease in this region. It is strange that we have no evidence to show that any part of the intermediate path between the organ of hearing and the auditory cortex may be a cause of such sensations. We should, indeed, hardly expect them to be due to a morbid process in the nerve fibres which conduct the auditory impulses, but the grey nuclei, in which the nerve fibres seem to end, might reasonably be thought capable of giving rise to impulses which produce, in the centre, the effect necessary for a sensation of sound. Yet such evidence is not forthcoming.

APPARENT LOCALITY OF THE SOUND.

Some important considerations are suggested by the common name of the symptom—"tinnitus aurium" or "tinnitus capitis,"—in popular language "noises in the ears" or "noises in the head." We may leave for the present the reference of the sound to the head itself. The question arises: Why is the subjective sound commonly referred to the ear and not to the external world? Subjective visual sensations always seem of outside origin, why do not those of hearing? The answer is complex, and brings before us many points connected with the subject. The apparent source of the sound seems to be, in part, a matter of mental inference. Continuous sensations—hissing or buzzing, for instance—seem as a rule to be of external origin when they are first observed, but their persistence soon convinces the sufferer that they cannot be. Knowing that they must have their origin in the ear he ceases to refer them to an external cause, and they then seem to him to be felt in the ear, although often, if he can separate the sensation and the inference, he can still for a moment realize that they seem due to something outside. It is so, also, with brief unfamiliar sounds. At first—now and then, indeed, always—they seem to have an external cause, but their repetition involves the knowledge that they have not, and the knowledge so acts on consciousness that they seem to be produced in the ear. An instance of this was afforded by a patient who was subject occasionally to the sound of a bell several times repeated. When he first became liable to the sound, in his annoyance, he sent a message to his next-door neighbour asking that the clock might be stopped, the sound of which so much distressed him, and he only discovered the subjective nature of the sound when he was informed that the neighbouring house did not contain a single striking clock. Having thus learned that the sound

could not be due to an external cause, it ceased to seem to be. An instance of persistent reference to an external cause was a girl subject to a sound exactly like two or three taps at the door; in spite of her past experience she frequently called out: "Come in." A woman who had long been liable to attacks of a rumbling sound, each lasting from a quarter to half a minute, was never able to distinguish them from distant thunder. Even after she had long known of their subjective nature, she would ask persons near her whether they did not hear distant thunder. Elaborate sounds generally seem external.

Pulsating Tinnitus.—On the other hand, pulsating tinnitus is almost always referred to the ear, even from its commencement. It may be compared by the patient to machinery and the like, but only as regards its character. The mechanism of pulsation in tinnitus is a question of great interest, because this feature is common when there is no increase in arterial pulsation and when there is no auditory hyperæsthesia. It is an instance of mechanical influence on nerve function. But the pulsatile element is not only a rhythmical interruption in the sound; there is often a character which is not purely auditory, although it is only from the observation of very intelligent patients that the fact can be ascertained. There is a local sensation beside that of sound; it is, perhaps, generally perceptible only as a local character of the sound; sometimes, however, it is distinctly a suggestion of a pulsating sensation within the ear distinct from that of hearing. This fact gives importance to other local sensations not auditory. One patient with labyrinthine deafness and attacks of vertigo had occasionally, as an isolated symptom, a brief sensation which could only be described as a "rushing out" through the ear, evidently much more a simple sensation than a sound. Another patient, a man aged fifty years, with changes

probably gouty, in the left labyrinth, causing some deafness, had a buzzing noise which would occasionally change to a throbbing sensation certainly not auditory; and sometimes there would occur behind the ear a momentary sensation as if the bone were being crushed in, neither pain nor sound. These cases show how much we need more facts on this point from patients with the ability to observe. I think that there must be sufferers among members of our own profession who are capable of adding much to our knowledge by careful personal observation.

Reference to the Ear.—It is not altogether difficult to understand such a feature as the local sensation I have referred to. We cannot doubt that nerve impulses are continually passing from the ear to the brain of which we have no consciousness. Disease of the internal ear may cause pain. Wherever pain may be caused—for instance, by inflammation—there must be afferent nerves, however free from sensibility the structure normally may seem to be. Wherever there are afferent nerves those nerves must be in constant activity, in some degree. In such an organ as the ear, morbid states which act on the auditory nerves may readily produce impulses also in nerves that are of different function—impulses that may act on consciousness. We can feel sure that unfelt afferent impulses are constantly produced in the middle ear. They must be continuously generated by its muscles. The action of at least one of these may cause a strong sensation, and the fact can be verified without difficulty. In many individuals, probably in all, a voluntary contraction can be produced in, apparently, the stapedius muscle. By closing the eyes tightly, and then trying to turn the eyeballs up, a loud fluttering sensation is produced in the ear, which continues as long as the contraction in the face lasts. It is in part a sound, but in greater part a simple sensation, evidently

the result of muscular contraction. A little practice will enable those who are sufficiently curious to produce it very readily by a mere contraction of the orbiculares.*

The nerves through which this sensation is produced must be constantly excited, without sensation, by the changes in the state of the muscles, which, we cannot doubt, are constant. A similar fluttering sensation may be a morbid symptom. A woman aged fifty-three was liable to occasional attacks of severe neuralgic pain in the left side of the head, and when the pain was at its height a fluttering in the left ear could be perceived lasting five or ten minutes at a time. Moreover, she presented an illustration of another association of these sensations; during the severe pain she would sometimes hear an occasional loud noise referred to the front of the ear and compared to guns going off. There is also some evidence to show that a spontaneous contraction in this muscle during sleep is the cause of the sensation of falling from a height, with which a dream is usually associated. The sound may be distinctly heard if there is premature waking.

I shall have presently to refer to the sounds that are referred to the head, but I may anticipate this to mention a very curious instance of another sensation not quite auditory. If we conceive the function of the cochlea as blending into tone, by repetition, the quickly recurring waves that are rhythmical, it is a curious question how we should hear such musical notes if the cochlea were absent. If the waves had sufficient amplitude, sufficient impetus, and were not too

* In passing I may note that if it is due to the stapedius, which is supplied by the same nerve as the orbicularis palpebrarum, the facial nerve, there is this interest in it that the function of the stapedius is believed to be to guard the labyrinthine structures from undue shock, by instantly contracting when sudden pressure tends to force in the membrane to which the stapes is attached, while the office of the orbicularis is, of course, to guard the eye. Thus each of these muscles which act together is the guardian of the organ of sense.

frequent, they would be perceived as isolated sounds, as are the vibrations by the sense of touch. Indeed, a tuning-fork vibrating twenty times a second is thus perceived if held near the ear, the vibrations being too distant to be blended into a tone. A very intelligent woman, after the cessation, under treatment, of paroxysmal headaches, became liable to a musical sound which seemed to fill the head and was accompanied with a strange feeling of extreme fulness of the head. Her hearing was perfect to all tests. She could hear Galton's whistle up to 16,000 vibrations a second. External noises prevented the subjective sound from being noticed. On sounding various tuning-forks she identified the sound as the middle C (between the treble and the bass). But the sound was always immediately preceded by a distinct sense of vibration in the head, which would sometimes remain as such, and at other times change to the sound. When she touched the sounding tuning-fork with her finger she said the feeling was exactly the same as that of the vibration she experienced in the head. I give this curious fact without comment. I believe the sensation was accurately described, and that the observation may be trusted. It is possible, perhaps probable, that the sensation was of central origin, but there must be a correspondence between the central action and peripheral excitation; sensations precisely such as are excited from the periphery may conceivably be of purely central origin. A similar sense of vibration is met with in other cases, although not frequently, and its associations deserve careful study. A woman, aged 50 years, had labyrinthine deafness, slight in the right ear, and moderate in the left, with slight continuous tinnitus, for many years. She had also had three attacks of more considerable tinnitus in the preceding four years, apparently in both ears, the second of which was said to have been accompanied with deafness, and yet with peculiar sensitiveness to sound, and to have ended

soon after a great increase in the tinnitus. The last attack had existed for six months, and was characterised by a frequent sound in the head, either general, or more on one side or the other. It was usually a very rapid vibration, but at times had been like music, although not any definite tune. Yet if she heard a tune played on the piano she would often continue to hear it after the instrument had ceased. This case also illustrates the association of cephalic vibration with musical tone. It also illustrates the apparent co-operation of the centre in the effects of labyrinthine change.

Reference to the Head.—These two cases are examples of the point next to be considered, the reference of subjective sounds to the head and not to the ear. The second case conforms to an explanation often given—that subjective sounds, produced simultaneously in both ears, seem to the sufferer to be in the head. This is occasionally true; but exceptions are, I think, more frequent. Bilateral tinnitus may be referred solely to the ears, and sounds may be referred to the head which seem produced only on one side. We see in this, moreover, the difference between abnormal local excitation of the nerves and their normal stimulation; when the latter occurs on both sides the sound is always referred to an external cause.

Sensations produced in one labyrinth are sometimes referred only to some one part of the head on that side. In one case of left-sided labyrinthine deafness, the sound was always referred to the left side of the head, but it seemed to spread when loud through the whole head; it was never referred to the ear. In other cases I have met with, it has been referred to the parietal region, to the parietal and occipital, to the occipital only, and in one, with considerable nerve deafness, a persistent rumbling sound was always referred to the region between the temple and the vertex, never

to the ear or any other part. In these cases of one-sided head-sound the extension to the whole head, when the sound becomes louder, is very common.

It is not easy to suggest an explanation of these varieties of localisation. We may, however, remember that the sound of a tuning-fork, applied to any part of the head, is referred, approximately, to the region on which it is placed, quite apart from the sense of vibration communicated to the bone; although many or even most waves must reach the labyrinth by the membrana tympani and ossicles, they pass also from the bone to the membranous labyrinth, either directly, or through the perilymph. The correct localisation of the sound must be due to the precise group of nerve endings that are chiefly stimulated. Since the causes of labyrinthine tinnitus are for the most part random processes, the nerve endings may be affected in various combinations, and it must happen that sometimes these combinations are such as would be excited from some part of the skull. The same subjective localisation will then occur. The stimulation of the nerves of ordinary sensibility may help to guide the localizing discernment. Yet cases are met with which seem to baffle all attempts to explain the apparent localisation. A woman with slight bilateral nerve deafness, who was unable to hear high notes, and on the right side could not hear any tone above C^3 (2112 vibrations per second), described a continuous squeaking sound, which was not referred to the ears, but seemed to her to be inside each parietal bone and to be louder on the side on which hearing was least affected. The subjective sound in this case corresponded nearly to that which the patient was unable to hear—a noteworthy instance of the double effect of local disease in hindering and causing sensation.

Localisation of Central Sounds.—It is a curious fact

that the subjective sounds which originate in the auditory centre in the cortex are referred to the same seats as those of labyrinthine origin. This is frequently the case with the central sounds included in this survey—those that occur as the warning of epileptic fits. When these are elaborate they are, it is true, referred to the external world, but when they are simple, or "crude," they generally seem to the patient to be produced either in the head, or in one or both ears. As examples I may mention a whistling sound referred to both ears, a similar sound referred to both sides of the head above the ears, a buzzing sound which seemed to pass through the head from one ear to the other, a whistle referred to the ear on the side on which the subsequent unilateral convulsion occurred, and a whistle seeming at the top of the head in one case and in another at the occiput. To the character of these epileptic sensations I shall have to return.

CHARACTERS OF LABYRINTHINE SOUNDS.

Some of the characters of the sounds that are due to labyrinthine disease have been already referred to, and others will have to be mentioned in connection with points yet to be considered. These variations are so great as to baffle any attempt at minute classification, as they also baffle the capacity of language. Our vocabulary, however extensive, is quite inadequate to describe our sensations, and the similes to which the sufferer has recourse are often misleading. A rough division may be made into (1) crude sounds, such as hissing, humming, machinery, rumbling, and the like; (2) tones, as a whistle, a simple musical note, or the sound of a bell; and (3) elaborate sounds, such as music or voices, distinct or indistinct. We cannot usefully separate the continuous and pulsating sounds, because a continuous sound so

often becomes pulsating when it is louder, but the pulsating character is chiefly confined to sounds of the first class. It is probable that the difference is of significance when invariable —when a sound, for instance, remains continuous, however loud it at times becomes, or remains pulsating however slight it may often be. The precise character of sounds needs to be carefully noted, since it will probably prove to be important, when we obtain more careful and discriminating observations.

Relation to Audition.—One feature which promises occasionally to be of definite practical importance, is the relation of the tinnitus to external sounds. In the majority of cases the subjective sounds are heard most when there is silence, sometimes are only then perceived. Doubtless external sounds often merely prevent notice, although we cannot be so sure that this is all. There are cases in which external sounds increase the tinnitus, and some of the facts are both curious and noteworthy. There may be a peculiar hyperacusis, by which certain sounds seem especially loud and unpleasant and increase the subjective sound. Although this feature suggests central co-operation the cases present evidence of labyrinthine change. Yet such co-operation seems the only way to explain another symptom met with especially in this connection, the occurrence of an echo or repetition of the sound, not always in the same pitch. A man aged seventy years, with slight reduction of hearing, not equal on the two sides, experienced an echo with musical notes and high pitched voices; every syllable seemed repeated in a lower note, although of the same duration. I found that the sound of C^1 tuning-fork and higher notes were thus repeated, but the middle C was not.* It is difficult to explain the change of

* The system of nomenclature of unusual notes employed in this lecture and the reasons for its adoption are fully explained in an appendix.

pitch as of labyrinthine origin. But in one case of the kind the labyrinthine affection was secondary to disease of the middle ear two years before, with lasting perforation of the tympanic membrane. A rushing sound was varied by buzzing and by an occasional sound like the ringing of several bells. Loud sounds seemed to "pass straight to the middle of the head"; some notes of the piano were particularly distressing, and the sound of these was repeated as an echo.

Allied to this symptom is another—the addition of an abnormal quality to sounds that are heard. For instance a peculiar clanging character was added to all sounds in a gouty patient thirty-four years of age, who had labyrinthine deafness and also suffered from other forms of more simple tinnitus. This may be conceived as labyrinthine, but it is often associated with a sense of discord and with increased sensitiveness, hyperacusis, which may be either peripheral or central. It is often very difficult to say to which we should ascribe the origin of the sense of discord. All music sounded discordant to a woman aged thirty-seven, who had slight double labyrinthine deafness, not equal, with simple tinnitus on the side of better hearing. On this she could hear the higher tuning forks (C^3 and C^2 and C, in the treble and below) better than the lower (C^1 above the treble), but she could not hear any note of Galton's whistle. These symptoms suggest that the discord was peripheral. But she also sometimes heard a sudden, spontaneous sound, as if some one had spoken to her, and the impression that this was a fact was strong for a moment, although she could never distinguish the words—a symptom which can hardly have been other than central. The production of a morbid functional state of the centre in consequence of the impulses that reach it, a state that involves the addition of sensations of central origin to those of labyrinthine source, is a question of some importance with regard to treatment.

Simple hyperacusis is not common. In one case in which it was partial, but co-existed with slight deafness, the sounds which were perceived were heard too loudly. Again a highly cultured man, aged fifty-seven, had slight nerve deafness on both sides, with tinnitus chiefly on the left, seldom on the right. It was like "gas escaping," and he had an occasional noise in the head, described as a "churning." But a moderate sound was heard too loudly; water poured into a bath sounded to him like the noise of a waterfall, and the voice of a person speaking seemed not only unduly loud, but also confused, which made it difficult for him to distinguish the words, in spite of their loudness. It caused a distressing sense of mental strain, such as is met with sometimes in other cases. The strain seems due to the effort needed to perceive what is heard, suggestive of some degree of cerebral difficulty analogous to word-deafness.

I may also mention the not uncommon cases in which all the senses are morbidly acute, in which, without sign of local disease, a combination of pulsating tinnitus and hyperacusis is met with; the arterial pulsations affect the nerves of the ear so as to influence consciousness.

The threads of this subject interlace in such a manner that I have been led away from the special point—the induction of tinnitus by sound. It is not often well marked, especially in such tinnitus as can be reasonably regarded as labyrinthine; but the important point, in such cases is the beneficial influence of silence on them. A clergyman, aged 54, certainly gouty, began to suffer at the age of 50, from gradual deafness of the right ear, tinnitus, and attacks of vertigo. An aural surgeon found the meatus and middle ear to be normal. The deafness was almost complete to high notes, C^1 (within the treble) being heard, but not C^2; the voice, however, was heard fairly well. The subjective sound was continuous in character, but it was slight or absent

when there was external silence, and was excited by any loud sound of more than brief duration. A short ride in a cab would make it distressing, although at starting he was free. The effect of the music in his church was so great that for a time he had to abstain from duty, and the effect of his own voice, if he read aloud for an hour, was almost unendurable. Either influence would cause the sound to become such that he compared it to "the blowing off of a full head of steam in a locomotive." Even then, if he retired to his room, in perfect silence, the sound would gradually lessen, and in the course of two hours would cease entirely. In this case great benefit was obtained by following the indication thus afforded, and securing prolonged freedom from external sound. In another case of increase by external sounds, the tinnitus was also continuous, but there was no defect of hearing, even to high notes, only an inability to discern "cross conversation."

Relation of Tinnitus and Vertigo.

Many of the cases of aural tinnitus, which have furnished the ground for these remarks, were brought under observation by the vertigo with which it is so frequently associated. But this common result of the irritation of the labyrinthine nerves is only within our survey so far as the subjective sounds are related to its occurrence. We refer the vertigo to the coincident affection of the semicircular canals by the same morbid process which, in the cochlea and vestibule, gives rise to the subjective sounds. That we are justified in doing so by the definite facts we possess is, I think, certain. The relation of the subjective sounds to the giddiness is, however, extremely variable. In current descriptions of aural vertigo it is often said that the noise becomes suddenly and rapidly louder to a culmination with which sudden intense giddiness coincides. This relation is quite excep-

tional. Such increase of sound may occur and culminate, for instance, in a sudden crash, without any attendant giddiness. But a gradual increase often precedes the vertigo. In the case of the clergyman just mentioned the increased loudness of the sound was often followed by an attack of giddiness, until he learned the value of external silence. He found that when this had diminished the subjective sound he was safe from an attack of vertigo. This is a significant fact, although not one to be too hastily interpreted. It seems to show that there is a certain solidarity, so to speak, in the labyrinthine functions for which we should scarcely be prepared by the difference in their apparent character, although it agrees with the continuity of the labyrinthine structures. Moreover, we can trace a like solidarity in the corresponding central functions, as we shall presently see. In other cases the tinnitus exists only just before the vertigo, although without any increase or culmination. In a man aged 36, with labyrinthine deafness on one side only and attacks of severe vertigo, the sound occurred only for two or three minutes before each attack of giddiness. It seemed to him to be in both ears and was compared to a very loud roaring, without pulsation. But the sound may simply coincide with the giddiness and not precede it. This was the relation in several cases, with evidence of one-sided labyrinthine change, causing deafness, in which the sound was pulsating and was compared to machinery, a steam-engine, and the like. In another case, of a clergyman aged 38, a continuous hissing sound occurred only during attacks of giddiness, which were accompanied by nausea and sometimes by retching. The sound and the vertigo always began together and the sound ceased as the vertigo gradually passed away, in the course of a few hours. The sound was referred to the left ear, but no permanent deafness could be discovered; yet as long as the noise lasted, in spite of the fact that it was

not very loud, he had great difficulty in hearing; a strong voluntary effort was needed to enable him to discern the nature of external sounds. I have already mentioned this peculiar difficulty, needing a mental strain to overcome it, as probably a central symptom, and not rare. In some other cases with definite one-sided labyrinthine deafness, a pulsating sound, compared to machinery or a steam-engine, was also noticed only with vertigo. It is not easy to explain the sudden paroxysmal effect of a process that is gradual and perhaps stationary. We have a similar phenomenon in the paroxysmal lightning pains of stationary tabes. These may occur during years, although the morbid process seems unchanged. These tabetic pains are perhaps a more pertinent analogy than the sudden attacks of pain in neuralgia, because we have good reason to refer the pains of tabes to the changes at the extremities of the sensory nerves, to which also our knowledge leads us to refer the attacks of tinnitus. Doubtless, causes which we sometimes can perceive, but more often fail to discern, determine the attacks, just as changes in the weather have so potent an influence in determining the pains of tabes.

SOUNDS OF CENTRAL ORIGIN.

Let us leave the ear itself and turn to the centre to which its impulses pass. As I have said, in the symptoms of epilepsy we can feel reasonably sure that we have to deal solely with the features of central sounds, although we cannot limit them to that disease. These higher auditory sensations vary very much in character; they may be crude or elaborate. I must pass by the subjective sensations which have a mental character, the auditory hallucinations and illusions, and those warnings of epilepsy which consist of a sense of spoken words. To touch upon their relations would carry us too far, and away from those with which we are specially concerned.

They have to do with instability in the higher functional regions of the brain, as is illustrated by the fact that epileptics with such an aura are in far greater danger than others of becoming insane. The cruder auditory sensations which usher in a fit, severe or slight, deserve careful consideration.

One curious fact is the occasional association of a subjective sound with a sensation of definite vertigo, which by itself is so common a warning. In some cases the central discharge mimics pure aural vertigo in a way that is alike curious and suggestive, as well as puzzling. The difficulty that is caused in diagnosis is the greater because there are cases of aural vertigo in which the suddenness and severity of the disturbance produces actual loss of consciousness. These cases are quite distinct from epilepsy and yet the superficial resemblance is close. This combined epileptic aura is another illustration of the fact of which I have just spoken—the very close connection which exists in the central processes between the two functions of the labyrinth, and also between the two symptoms which its disturbance generates. The sound which accompanies the epileptic vertigo is as simple as that which occurs in the labyrinthine form. The association is the more remarkable because all our knowledge points to the motor centres as the source of the sensation of giddiness, while that of sound is purely sensory. After all, however, the connection seems to illustrate and emphasise the fact I have already mentioned—the fact that, since all the sensory impulses produced at the periphery are perceived through a related activity of the higher centres, the functional processes in these must correspond to every peripheral process, and may present similar associations.

Even the pulsatile character of aural tinnitus may be reproduced in the centre. It is unquestionably a rare symptom, but is of great interest. In a highly complex aura which

I have recorded elsewhere,* a sensation of beating in the chest passed up to the head and there became a pulsating sound, accompanied by two lights before the eyes, which approached with a rhythmical motion. We must, I think, ascribe this character to the mechanical influence of the arterial pulsations on the discharging centre. We know the influence of such pulsation upon pain; we know the susceptibility of nerve structures to mechanical influences, and we must assume that in the process of actual discharge, all their susceptibility is augmented. The influence is, therefore, not difficult to understand, whether the pulsatile pressure augments or hinders the progressive liberation of the nerve energy. It is not rare for the central sound to be described as "machinery," and this generally implies pulsation. But more rapid variations may give the sensation the character of vibration, a feature we must attribute to the character of the nerve action itself. We have seen the relation of such rapid vibrations to low musical notes, and the nerve processes for this relation must have a corresponding development in the brain. A vibrating sound, compared to the noise of a faradic apparatus, was referred to the left ear by one patient as the warning of left-sided fits. Here, again, we have in a very curious degree the central reproductions of peripheral impressions, because such a sensation sometimes apparently originates in the ear itself.

Although the elaborate sensations of psychical character, definite words and the like, are not within my subject, I may mention that in patients with such a warning of severe attacks, the minor attacks—*petit mal*—may be characterised by a quite simple auditory sensation. In one case of the kind simple "buzzing," referred to both ears, with obscuration of consciousness, constituted the slight attacks, while the onset

* In the preceding lecture on Subjective Visual Sensations.

of the severe seizures was characterised by a vision of persons who seemed to speak intelligible words.

Central Co-operation in Labyrinthine Sounds.

In leaving the higher centre we must, however, return once more to the labyrinth. A point that is not only of interest, but will prove, I think, of practical importance, is the co-operation of the centre in determining the character of the sounds produced by labyrinthine irritation. This is not difficult to understand when we consider how disturbing to the central structure must be constant abnormal impulses from the ear. The fact which raises this question is the frequency with which labyrinthine deafness is associated with sounds of such elaboration that it is very difficult to think that they are solely peripheral in nature. Another important question also is thus brought before us: What degree of elaboration of sound is consistent with a purely labyrinthine origin? Of course, the crude, or simple, sounds, which have occupied us so much, we must regard as peripheral. On the other hand, elaborate sounds, such as a voice uttering distinct, or even indistinct, words, must be regarded as central. But there are other forms of tinnitus that cannot be referred with confidence to the labyrinth alone. To it, indeed, we may reasonably ascribe all sudden, simple tones, when we consider the analogy of the brief, momentary sensations of pain which are experienced in tabes, and the grounds we have for ascribing them to the morbid action of the degenerated nerve endings. This applies especially to the sudden sound of a bell, which is not rare, and even to repeated bell-like sounds. But we must, I think, recognise a secondary central action in such a case as that of a woman, aged 43, in whom, after an attack of giddiness, bilateral tinnitus referred to the ears commenced and increased gradually. It was a buzzing sound, louder at

night. After a time there was an occasional change, for a few hours or days, to that which seemed to her just like a band of musical instruments. She described it as so distinct that, although she could not recognise the tune, she believed that if she had been acquainted with harmony she could have written down the full musical score. At times also she seemed to hear voices, and, although she never could distinguish words, the sound was that of a human voice, and occasionally she had seemed to hear herself called. But this was no hallucination. She referred all these sounds to the ears, and they never seemed to her, for a moment, to be of external origin.

There is much more that I should like to say that I must leave unsaid. I should like to have referred to some conceptions of the process of stimulation of the special senses, which might establish more unity in our thoughts, and with this to have considered the probable relation of the energy which excites the nerve impulse to that which is excited, and the nature of this. This subject is too wide for me to touch upon, but I would call attention to some other facts related to my special subject, although I can only mention them. One is the frequency with which labyrinthine tinnitus becomes associated with unpleasant cephalic sensations, feelings of pressure, fulness, throbbing and the like, constant or varying, and sometimes related to the sound in a way which connects them with the tinnitus that is referred to the head itself. Another subject is the relation of tinnitus to paroxysmal neuralgic pain, either in the ear or head—a fact of much suggestiveness that well deserves study. But these at least for the present we must leave.

PATHOLOGY OF TINNITUS.

It is strange how few facts we have regarding the pathological changes which underlie the symptoms in the common class of cases, with deafness, tinnitus, and often vertigo, gradual in onset and often slowly progressive. It is strange, because our poor-law infirmaries and work-houses contain always many cases of the kind; it is strange, also, because modern methods have increased so much the revealing power of the microscope, and because the present generation of workers are not prone to leave neglected any unexplored field. But we can surmise much with reasonable sureness. In cases with striking and unusual symptoms, conspicuous changes have been found, concretions and the like. Further, we know that the parts chiefly concerned are of fibrous and epitheliated structure, in which epithelial hairs receive the sound vibrations, which thus excite nerve impulses in the delicate structures by which the two are united. We know, also, with what extreme readiness this process must be deranged and how minute a structural alteration may suffice. We know, also, that this membranous labyrinth may be the seat of acute inflammations, of which the most significant is that which occurs in early life, and sometimes in adults, as the effect of a peculiar blood state, often excited by cold, one of those toxic conditions which have a special local incidence, for the inflammation is limited and symmetrical. It is now generally recognised that when there is complete deafness as the sole result of what was regarded as an attack of meningitis in early life, the malady was not meningitis but bilateral inflammation of the labyrinth, the general cerebral symptoms of which may closely resemble those of meningitis. We have in this fact, not only the indication of a special blood state, but also of special susceptibility on the part of the labyrinth. We know from many other facts that such susceptibility to

acute inflammations of this kind on the part of fibrous structures involves also a proneness to suffer from chronic changes in later life, especially in cases where there is an inherited rheumatic or gouty tendency, the tendency which may be called, and I think has been called, "tissue gout." We can thus understand the fact, so often illustrated, that these chronic labyrinthine changes are especially common in such subjects. We also know that in late life, apart from such predisposition, the tendency to analogous affections of other fibrous structures is common. We see the great tendency to chronic rheumatism presented by the old, which we may ascribe to the influence on the tissues of waning vitality, coupled with the effect on the blood of the age-imperfection of the organs on which the blood state chiefly depends. It happens that only a week ago a pertinent illustration of this came under my notice. A man aged fifty-eight years, with some family history of rheumatism, although not of known gout, had suffered from vertigo and for some years from tinnitus and from deafness. His tissue tendencies were clearly shown by a contraction of the palmar fascia of one hand, on the ulnar side, passing down the little finger and keeping it flexed. The deafness was partly labyrinthine, but chiefly due to the middle ear, and the report of an aural surgeon (Mr. Field) on the cause of this was very instructive. He found considerable thickening of the membrana tympani on each side with complete fixation of the ossicles. The latter must be due to fibroid adhesions, while the state of the membrane was doubtless an indication of a similar change throughout the cavity, including no doubt the membranes which close the foramina leading to the labyrinth. Moreover, the clear evidence of labyrinthine deafness, in addition to the impaired conduction, leaves little doubt that there was a like condition in the labyrinth, although perhaps in a less degree. We cannot doubt the influence of such fibroid thickening on

the structure in which the epithelial hairs are connected with the nerve endings. Morbid processes of other nature, syphilitic, traumatic, and the like, are also met with in such cases.

Primary Atrophy of the Auditory Nerve.—Very similar symptoms are, moreover, produced by a primary atrophy of the nerve, a subject to which I may briefly digress. I should like to utter an emphatic protest against the readiness to invoke atrophy of the auditory nerve as the morbid state whenever labyrinthine deafness is recognised in conditions in which nerve degeneration is sometimes met with, and especially in tabes. It is, indeed, partly for this reason that I have preferred, for the most part, in this lecture, to use the term "labyrinthine deafness" rather than "nerve deafness." Statistics have been published in which in every case of tabes with any degree of labyrinthine deafness, this impairment has been put down to atrophy of the auditory nerve; such atrophy has therefore been said to exist in a considerable proportion of the cases, and the facts have been received and quoted as conclusive. Symptoms of more or less random labyrinthine changes, generally non-progressive, are not rarely met with in tabes. The previous life of a large number of tabetic patients has been such as disposed them to tissue degenerations, and the effect of the blood state is often much increased by the diminished amount of exercise which the developed malady entails. Among all the cases—and I have careful notes of many hundred cases of tabes—I can count on my fingers those in which there was reasonable evidence of auditory nerve atrophy. Until, therefore, we have far more definite knowledge of the symptoms of primary atrophy, more caution should be exercised in the diagnosis, or even suspicion, of this change. But we sometimes meet with evidence of the process which cannot admit of doubt. A man with tabes, who had bilateral deaf-

ness, presented a progressive concentric limitation of the range of hearing quite similar to that which occurs so often in tabetic atrophy of the optic nerve. When first examined he was quite deaf on one side, and on the other could hear only the notes between G (within the bass) and E^2 (above the treble), the range of audition being thus little more than two octaves. In the course of the next month the contraction of the range had reduced it to one octave, from middle E (on the lowest line of the treble) to E^1 in the highest space, and so sharp was the limitation that while he could hear E he could not hear E flat, a semitone below; soon afterwards the deafness was total. But atrophy of the optic nerve does not always take the form of progressive concentric restriction, and it is not likely to be the only, and perhaps not the most common, manifestation of that of the auditory nerve. Atrophy of the optic nerve is so often progressive, when once established, that we are justified in regarding with suspicion all cases of alleged auditory nerve atrophy in which a progressive course is not conspicuous.

Functional State of the Receptive Structures.—It may seem strange at first that defective hearing should so constantly coincide with the production of these spontaneous sensations. But the due reception of external stimuli and their adequate effect upon the nerve endings must depend upon structural integrity, molecular integrity we may say, in the receptive elements. Organic changes in these may prevent the sound-waves producing their due effect, and yet may also induce the molecular alterations in the terminations of the nerves on which spontaneous sensations depend. We may remember how frequently the distressing pains of tabes co-exist with loss of sensibility to pain, and also the disproportion between the effects on consciousness and the processes on which they depend.

Another fact of at least equal importance. In the normal condition all nerve structures must be in constant functional activity—at least, in some degree. This is necessary for the maintenance of nutrition; it is a necessary effect of vitality; it is essential for the capacity for full action on an adequate stimulation. In the case of a large number of nerves we are quite unconscious of this continuous functional activity. It need be only of the slightest degree, to subserve that constant renewal of molecules which must occur for life to be maintained and function possible; but the phenomena of disease show us that a similar persistent action is also a part of the processes of life under abnormal conditions. It seems to be as constant when nerve endings have undergone slight degenerative changes as when they are normal. The morbid alterations in the fibrous tissue of the labyrinth, of which I have spoken, do not necessarily destroy the nerve endings, and they only interfere with their function in so far as the terminations are altered and the effect of sound-waves is hindered. We can thus understand that the subjective sounds are the result, under altered conditions, of the same vital state which normally maintains the nerve in perfect readiness for instant action.* We can understand that the damaged structure, and altered molecular arrangements, while they do not impair spontaneous activity, change its form and increase the energy of the impulses. It is not, I think, difficult thus to perceive that the persistence of these sounds, which seem so strange, especially when combined with deafness, is due to one of the conditions on which the functional capacity of the nerve structures essentially depends.

* Regarding this continuous activity see Professor Michael Foster's lecture in The Lancet of Nov. 7, 1896; also note by myself on "The Dual Activity of Nerve Cells," The Lancet, May 10, 1890. The words "nerve structure" should apparently be now used instead of nerve cells.

TREATMENT.

That which I can say regarding the treatment of these symptoms must of necessity be limited to general principles. Details I am obliged to omit, and if that which I say seems to be meagre, believe me this subject has been before my mind more definitely than any other. The treatment of these affections constitutes one of the most difficult and most obscure branches of therapeutics. This is not indeed surprising. We have seen that to a large extent they depend upon chronic processes. Of all slow processes of disease it may be said that the morbid process, at every period and at every stage, is an accomplished fact. Damage and cicatrisation go hand in hand, but the process of cicatrisation is not recovery. In acute disease, the alterations caused in the tissue may pass away to a large extent, and the normal state to that extent may be reproduced. But a change indistinguishable from cicatrisation perpetuates all slow disease, and thus its effects necessarily endure. Still, even over these, we are not quite powerless, and we learn, on every side, lessons which should teach us to be slow to give up hope, or to relax our efforts to obtain that which we now lack. How impressive is the lesson we have just had from science that the word "impossible" should find no place in our vocabulary. Even two years ago, had the wisest among us been asked the question: Is it possible that before the end of the century we may be able to see through a deal door or a human leg—what would have been the answer? The humility which this should teach should equally prevent us from limiting the possibilities of power in any department of our science or any branch of our art that is founded thereupon. The reason why, through this effort to discern some of the relations of those subjective sounds, their treatment has been ever before me, is because the first necessary step for the attainment of greater power

is more minute and accurate recognition of their features and relations. It is through these that we must hope to be able to apply, with more prospect of good, the measures which we already know to have some influence, and, by the discrimination they permit, to employ more wisely any means which the future may give us. Empirical measures, on which we are obliged so largely to depend in many other maladies, have at present almost no place in the treatment of tinnitus. We know, for instance, that the noises are sometimes lessened by bromide, although the influence of this is far less than on the vertigo which is so often associated. But we know also that the influence of bromide is especially exerted on the centres in the brain. I doubt, indeed, whether we have at present any evidence of its action on the peripheral nerve structures. But the evidence I have mentioned, that the auditory centres become secondarily involved in some cases, and give rise to some special features of the subjective sensations, is reason for a careful systematic attempt to discern the relation between such central co-operation and the power of bromide to relieve the symptoms. The subsidence of the sounds in silence, and the lasting relief thus afforded in suitable cases, I have already sufficiently dwelt on. There are many cases of labyrinthine tinnitus in which counter-irritation has a considerable influence for good, but there are also many cases in which it is powerless, and we are not yet able to distinguish the two. The distinction can only come, but probably will come, from careful discernment of the conditions under which the process develops and of the precise symptoms by which it is manifested. At present we cannot form any opinion regarding the significance of differences in the minute characters of the subjective sounds. We are already supplied with so many means of lessening the morbid action of other peripheral nerves that it will be strange if the future does not yield us means of

lessening, at least in some degree, that of the nerves of the labyrinth. We can find little trustworthy guidance from experience of individual sufferers. In one labyrinthine case, it is true, the sound was always lessened by firm pressure on the posterior part of the temporal region, and the fact deserves to be remembered. Another patient found that a diminution was always caused by inclination of the head towards the side on which the tinnitus was heard, but this, I think, is only a special example of a more frequent influence,—the sound may be often lessened by pressure on the arteries of the neck. Strange to say, this result has seemed to me more common when the tinnitus is a continuous sound than when it is pulsating.* I cannot now pursue the possible suggestiveness of this fact, but it leads up to another. I feel compelled to mention to you one case, which has been described in Germany,† on account of the importance of the lesson it conveys. An elderly man had gradually become completely deaf in the right ear, with most troublesome tinnitus. The left ear was becoming deaf in the same way. The tinnitus was so loud, so persistent, and so distressing that the man had several times declared that he could endure it no longer and must end his life This proceeding on his part was made unnecessary by the treatment which was adopted. It was found that pressure on the carotid artery diminished the sound. The right internal carotid was therefore tied. It is very difficult to understand the grounds for the operation. The blood supply to the labyrinth comes partly from the external carotid and partly from the basilar. The path by the auditory nerve to the opposite hemisphere is entirely within the blood supply of the basilar. The opposite auditory centre is supplied by the opposite middle cerebral artery. It is thus easy to under-

* The fact of reduction of pulsating tinnitus by pressure on the vertebrals has been described by Dr. Dundas Grant.

† Linsmayer, Wien. Med. Blätter, 1893, Nos. 8 and 9.

stand that the operation was found to have no effect whatever on the tinnitus. It produced left hemiplegia and left hemianopia, and death resulted on the fifth day. I draw attention to this case because I think it should forever prevent the repetition of this operation for the relief of labyrinthine tinnitus.

I must end this outline of some of the more prominent features of the subject, by reiterating the hope that it may at least serve to stimulate others to its careful study, and may serve to guide their observations. Provinces of medicine are often unproductive because they need more careful cultivation than they have yet received. We have to till the fields to which our path may lead us, however barren they may seem to be, and hope that some of the seed sown, even upon stony ground, may germinate and bring forth fruit in time to come.

NOTE.

THE DESIGNATION OF MUSICAL NOTES IN SCIENCE AND MEDICINE.

("Review of Neurology and Psychiatry," April, 1903.)

The convenient and accurate designation of musical notes has become a subject of importance to medicine and to science, especially to physiology. The system current among musicians has come down from the early days of the organ,* but it was adopted by Helmholtz and has therefore been used by most writers, since the publication of his classical work on "Tonempfindungen." But it is a system which has no rational foundation, except in organ construction, and with this it has only a partial connection. The absence of intelligible ground for it has had the necessary consequence of mistakes by those who have attempted to apply it to wider needs. It is not too much to say that error and confusion are met with as frequently as precision.

The need for precise designation occurs in describing normal hearing, and still more in recording the limitations of hearing met with in disease. The latter is as frequent in the work of the physician as in that of the aural surgeon. In the various senile and gouty changes in the internal ear, so often associated with vertigo, in the results of other forms of ear disease and central changes, and in the atrophy of the auditory nerve sometimes met with in tabes, there is often peculiar restriction of the range of audition. The method of designation by means of the number of vibrations per second is not convenient except for the higher notes, above the range of the tuning fork. For lower

* It originated in the sixteenth century, by a development of an earlier system, and was due to the extension of the notes of the organ. See Rockstro's Art., "Tablature," Grove's "Dict. of Music."

notes the musician's method of designating the notes by letters and distinguishing by numbers the successive octaves is far better, and is generally employed.

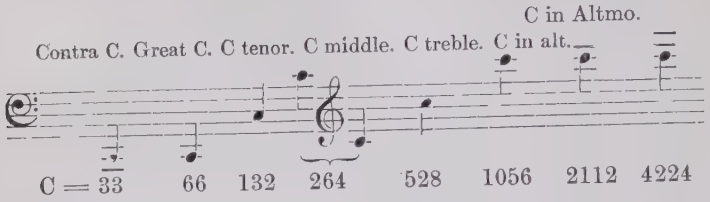
The term "octave" is in universal use as a name for each series of seven notes. It is apparently due to the fact that the eighth note is in unison with the first, although it really belongs to the next "octave." The more accurate term "septave" has been occasionally employed, first nearly a hundred years ago,* but few musicians have ever heard it. Since the series of musical notes, without added sharps or flats, has C for the keynote, the system of making each designated octave begin with C is universal. Each note of the series of seven being indicated by a letter, the successive series or "octaves" are distinguished by a number, or by strokes, after each letter, above for the higher, below for the lower. Instead of strokes or numbers, the method of doubling or trebling the letters has also been employed.

To distinguish the higher from the lower, some starting-place is necessary. The great source of confusion is that, in the current system (or what is supposed to be such), the starting-place for the higher and lower octaves is not a singly conveniently placed unnumbered octave, but is constituted by two octaves, both unnumbered, inconveniently placed, and distinguished by a difference in the letters that indicate the notes. The upper one is indicated by small letters, the lower by capital letters. Each is unmarked, but the former is called the unmarked octave (the "unstroked," "ungestrichen" octave, because strokes were used of old instead of letters); the latter is distinguished as the great octave, apparently from some relation to the "great organ." The "unmarked" octave extends up from the C in the middle of the bass, the great octave from the C on the second leger-line below the bass.

This method will be better understood if the designations are added to the note-symbols as in the following table. It must be remembered that each octave extends upwards from the C to

* In a rare little book on "The Art of Tuning," by Earl Stanhope, Lond., 1806. A copy is in the Library of the Athenæum Club, London.

the B above. I have added to it a serial indication of some of the errors which are to be found in scientific and medical writings.



	Contra.	Great.	Unmarked.	One time marked.	Two times marked.	Three times marked.	Four times marked.	Five times marked.
1. Helmholtz,	C_1	C	c	c^1 or c'	c^2 or c''	c^3	c^4	c^5
2. Tyndall,	C^1	C	c	c^I	c^{II}	c^{III}	c^{IV}	c^V
3. Bosanquet,	C or ,C	C	C^0	C^1	C^2	C^3	C^4	C^5
4. Banister,	CC	C	C	c	c	c	c	c
5. Peterson,		C_1	C	c	c′	c″	c‴	c⁗
6. "Acoustics" Enc. Brit.,				C	C_1	C_2	C_3	
7. Foster,	C_1	C	C	C^1	C^2	C^3		
8. M'Kendrick,	Do_1	Do^1	Do^2	Do^3	Do^4	Do^5	Do^6	Do^7

1. Helmholtz, "Tonempfindungen," dritte Ausgabe, 1870.
2. "Lectures on Sound," quoting from Helmholtz.
3. Encyc. Brit., art. "Music," part II. The capitals for the upper notes are about one quarter smaller than those for the lower.
4. "Handbook of Music." The double C for C_1 is open to mistake, although really clear.
5. Art. "Harmony," Chambers's Encyc.
6. Author's initials only given. The actual note adduced is A. "A_3 is three octaves above the A between the second and third lines of the treble clef."
7. Sir M. Foster's "Manual of Physiology," all editions. Only the upper and lower notes are given, but the series follows from the vibrations assigned to them.
8. "Physiology of the Senses." "Fa" is the note given, but from the vibrations assigned to it in the several positions, the above series follows with no unmarked octave.

In Politzer's "Handbook of Diseases of the Ear," Eng. trans., Cassells, 1883, the same note, C = 512, is referred to on page 167 as C^3, and on page 178 as C.

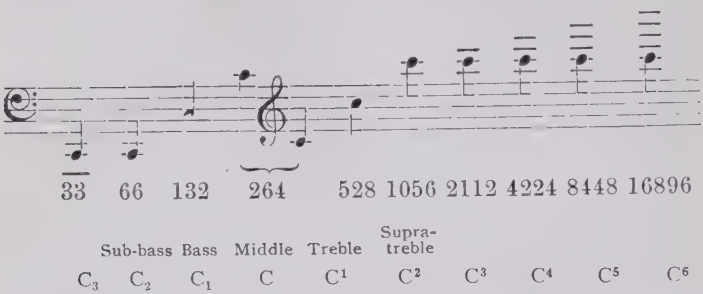
More examples might be adduced, but these suffice to show the confusion that has resulted under the present system of two unmarked octaves, in an arbitrary situation, for which no clear reason exists to help the memory or to induce uniformity. The

distinction of capitals and small letters, the former for the few octaves below the C within the bass, and the latter for it and the numerous octaves above it, has evidently been felt by some writers to be an anomaly not worth conformity. This seems to have been the opinion of Grove, since in every article in his "Dictionary of Music" similar capitals are employed for the high and low notes, with the distinction of some descriptive designation.

It is not worth while to discuss in detail the examples of variation I have given. Most are simple mistakes, as their examination will show. I have taken pains to ensure precision in every example. It is certain that in Science a more simple and a rational system is needed, if uniformity and exactness are to be secured. It seems also that in Music the need is scarcely less. There is no sharp division between Music and Acoustics, or between the latter and Physiology.

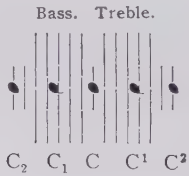
For a useful system it is essential that there should be only one unmarked octave, or "neutral octave," as I think it might be conveniently termed. The best position for this is certainly the octave above the "unmarked octave" of Helmholtz and the Germans. This position is assumed in the article on "Acoustics" in the Encyc. Brit., and, for the higher unmarked octave, by Peterson ("Harmony") in Chambers's Encyclopædia. Its advantages have been strongly urged by Dr. Shinn, of the Guildhall School of Music, in a letter to the "Musical News," January 24th, 1903, in the pages of which I had endeavoured to evoke some discussion of the subject. I first suggested that the higher of the two present "neutral octaves" would be most likely to obtain adoption, but the advantage of the octave above this is manifest. The neutral C is then that which is often fitly called the "middle C," between the bass and treble staves; C^1 and C_1 are each within the treble and bass staves respectively; C^2 and C_2 are each on the second leger-line, below and above the respective bass and treble, and may be called the sub-bass and supra-treble. These words are a convenient and sufficient distinction in oral description. It is not only desirable, but necessary, to secure precision and accuracy, that a consistent and reasonable method should be employed.

The following will then be the method that is suggested:—



Tuning forks avail us up to C^3, but the range of hearing extends, in young persons, at least three octaves higher, and for these upper notes Galton's whistle is available. It is an instrument of the greatest service in the estimation of the function of the auditory nerve.*

The symmetry of the suggested method will be more clearly perceived if the staves are placed vertically.



Moreover, the system aids the memory of the number of vibrations of these several notes. The bass C_3 is 33 vibrations. The first (leger-line) C below the bass (C_2) is the first with two figures (66), and the first above the treble (C^2) is the first with four figures (1056), while the number of vibrations of C^4 (4224) is almost as easily remembered as C_3. This may be a trifle, but it is a conve-

* It is less known than it deserves to be. It is made by Hawksley, at the suggestion of Mr. Francis Galton. It may be well to mention to those who use it that it should be blown through from the mouth, not breathed through from the lungs, to prevent the interior of the fine steel tube becoming rusted. For the same reason it is well to draw air through the tube after use.

nient trifle, and I have yet to learn that even that justification can be urged in favour of the old method, venerable as it is, and familiar as it is to many.

I may be permitted to end this note with one example of the need for uniform precision. In a book, published in 1885, I wrote this: "The patient is absolutely deaf to the loudest musical notes above E of the treble clef and below the lower G of the bass." The range of hearing afterwards lessened, "until only the notes between the two E's of the treble clef can be perceived." In a German translation* the passage is rendered: "für die lautesten musikalischen Noten über E und unter contra-G völlig taub ist." . . . "Nur noch die zwischen E und E legenden Noten wahrgenommen werden." I may say that the G I intended to indicate was that on the lowest line of the bass clef, an octave above the "contra-G."

* "Gehirnkrankheiten," von W. R. Gowers, ubersetz. von J. Mommsen, 1886.

LECTURE III.

ABIOTROPHY; DISEASES FROM DEFECT OF LIFE.

*Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, Bloomsbury, on February 21, 1902.**

Gentlemen:—Life presents to us a double aspect, and so therefore does death. One is its somatic character, the life of the organism as a whole, to which the words "life" and "death" are chiefly applied. Somatic life depends essentially upon the blood, as familiar words assert, words uttered and written long centuries ago—"the blood which is the life thereof." It depends on the proper constitution of the blood, deficiency or perversion of which will end life speedily or slowly. It depends on the due reception of oxygen by the blood through the lungs. And it depends also, and most of all, on the due movement of the blood by the heart; if the heart is stilled, life ends almost instantly. But besides this general life, the termination of which involves that of every part of the body, many of these parts have their own vitality. Some of them may slowly die, while the life of all the rest goes on without impairment. They may die from many causes, some early, inevitably, from a grave defect of vital endurance; some much later, the failure being but slightly premature; and some at various times, apparently from various causes. When the failure is early it is often due purely to a defect in vitality, a defect which seems to be

* The Lancet, April 12, 1902.

inherent, the tendency thereto inborn. We do not, indeed, apply the word "death" to this slow decay of the elements; we speak of it as "degeneration," but the process is in many cases, perhaps in most, an essential failure of vitality, and I think it is instructive to consider the degenerations in this aspect. But in doing so I am met by the difficulty, that we have no word by which to designate this conception—a degeneration or decay in consequence of a defect of vital endurance.* I do not like new words—indeed, I dislike them—but if we have a conception for which no name exists, which we need frequently to speak of, it is not wise, I think, to shrink from an attempt to give it a name. Here, the simplest mode of obtaining what we need is to insert the root of *βιος* after the negative particle in "atrophy"; this gives us "abiotrophy." But it is generally better, if you can, to appropriate what you need than to make it afresh, and we find the word *βιοτροφος* used in the sense of "vital nutrition."† If we prefix the negative particle we have the same word, "abiotrophy." If a more general term is desired, the adjective "biotic" has been occasionally used in English ‡ in the sense of vital, and from this we may form "abiotic" to designate that which depends on defective vitality. A corresponding substantive would be "abiosis," and this also has some war-

* This conception of the degenerations of the nervous system was admirably stated by Dr. F. W. Mott in his Croonian Lectures for 1900. But it formed the subject of an unpublished lecture which I gave in 1897 or 1898, and is epitomised, in its essential features, in some remarks which I made at a discussion on Tabes at a meeting of the Pathological Society of London, on December 5, 1899, reported in the Lancet of December 9, 1899, p. 1591.

† Not indeed in classical Greek: it is quoted by Stephanus from Gorgias Pisides, a Byzantine writer of the seventh century (620 A. D.).

‡ First in 1600. Dr. W. B. Carpenter added a second adjectival termination and made it "biotical." It exists in French as *biotique*, from the Latin *bioticus*, and the Greek *βιοτικος*, from *βιοτος*, and *αβιωτος* had the sense of "lifeless." Thus, "abiotic" is clearly legitimate.

rant. The word βιωσις was employed once or twice in old Greek, meaning "mode of life." It is given us by the physician, who is more widely known and esteemed, on account of the character and precision of his writings and the devotion of his life, than any other medical man who has lived—St. Luke. It is pleasant, I think, to take a word from him.* So I give you a choice of words by which to designate this unnamed conception.

Cutaneous Abiotrophy.

In most gatherings of men we may see an illustration of abiotrophy. We may perceive an illustration of the failure of the life of the hair follicles of the scalp which must be the essential cause of early baldness. We are so familiar with the condition that we do not always perceive the lessons it may teach us. In many men most of the hair follicles die soon after adult life is reached—die beyond the power of restoration. Some, however, persist; around the circumference is a zone in which vitality is retained; elsewhere the failure is absolute and uniform. It is certainly often hereditary; father and son—indeed, also grandfather and grandson—may become bald between the ages of 20 and 30 years. Such decay is practically limited to the male sex. In other persons and families failure of vitality in the follicles occurs later and is less extensive. In others, again, a luxuriant growth of hair is retained throughout life. I know that baldness has lately been ascribed to seborrhœa, due to organisms, but such a cause, accidental and acquired, fails entirely to account for the more general features of the condition, and, if true, can

* The word, previously used in the Septuagint, comes to us through the author of the Acts of the Apostles, but may have been used by St. Paul, since it occurs in his speech before Agrippa.

be only an exciting cause, effective by reason of the failure in vitality.*

Without complete degeneration of the follicles they may fail to produce the normal pigment of the hair, and early greyness is hardly less common than early baldness. It is not less instructive. It is a qualitative failure, an enduring defect of one function of the follicles. It shows that without the total failure of life part of the work of life may come to an end. That it must be by a chemical process that the pigment is produced I think none can doubt, and that hair may be blanched by a change in the follicular secretion is conclusively shown by the case of one-sided greyness of the hair of the head and beard produced in a few days by meningeal hæmorrhage.† Regarding the association of greyness and baldness, not only may the imperfection to which greyness is due be extreme without any tendency to loss of hair, but it is said that baldness is seldom preceded by greyness. The persistent luxuriance of growth of the hair when the formation of pigment has ceased is a familiar fact. It is also worthy of note that even early greyness is met with in each sex, and does not present the tendency to affect males only, which is so conspicuous in premature loss of the hair.

Muscular Abiotrophy.

I must not be tempted to delay too long at the surface of the body, but we must not pass at once to the nervous system. Between the surface and the nerve centres we meet with tissues which present very striking and instructive forms of true abiotrophy—the muscles. In the various forms of

* In curious contrast to the seborrhœal theory is the conclusion of one German writer that early baldness is the result of the habit of daily washing the head.

† Described in the following lecture on "Metallic Poisoning," p. 153.

idiopathic muscular atrophy, in which there is a primary atrophy of the muscular fibres, we have examples of a true abiosis. To all these primary myopathies it has become customary to apply the term "muscular dystrophy," and the custom is convenient, if not quite accurate. The term thus includes both simple muscular atrophy and its well-known congener, pseudo-hypertrophic paralysis. In these the muscular fibres, after full development, cease to maintain their nutrition. They slowly waste and a large number, most of them in many parts, all in some, ultimately perish. The connective tissue between them overgrows by a process we can discuss better in connection with the nervous system. Its increase may fail to maintain the normal bulk of the muscles, so that these waste conspicuously, sometimes extremely. In other cases this tissue-weed, as we may regard it, presents a more luxuriant development, and produces fat-bearing cells which so much increase the bulk of the muscle as to cause the enlargement of "pseudo-hypertrophy."

These great variations in the interstitial process compel us to regard the failure of life in the muscular fibres as the essential element of the disease; it is a defect of vital endurance, truly congenital in so far as the tendency is concerned. Two other facts indicate the same conclusion. The defect in the muscular system may not only be, as it were, qualitative, it may be also quantitative. Certain muscles may be absent, however early the sufferer is examined, especially the lower part of the pectoralis major and the latissimus dorsi, which depress the raised arm and are the least needed muscles of the body, since gravitation will accomplish in most circumstances that for which they are needed. We have further proof of its dependence on a congenital tendency in its occurrence in several members of the same family and chiefly in the males, the girls, who do not suffer, conveying the morbid tendency to their own sons. Girls, indeed, may suffer, but

less frequently in affected families than in the isolated cases which we sometimes meet with. Remember that all hereditary and family diseases sometimes arise *de novo*. I shall have to return to the point again, for it is important.

We saw the special tendency of males to suffer from baldness, we shall see the tendency also in the affections of the nervous system; its manifestation in pseudo-hypertrophic paralysis is striking—even more so, I think, than in simple idiopathic atrophy. You may have heard of the group of cases through which Dr. Meryon first made the disease known in this country. Four boys, all the sons in one family, died from it; the girls were unaffected. But I know that one of these has two sons, and both are affected in an extreme degree. Her daughters are quite well. I could give you other examples which have come under my own observation which are equally marked, but the fact is too well known to need further illustration. I will only remind you, before passing from these muscular maladies, of the unequal way in which the muscles suffer. The distribution of the affection is strikingly different from that of those atrophies which depend upon the nervous system, but its localisation varies also in the different varieties of muscular dystrophy we are able to distinguish.

Abiotrophy in the Nervous System.

Let us pass to the degenerations of the nervous system. In all those with which we are concerned to-day there is a slow decay of the nerve elements which have a common function—a decay limited to these but extending through their entire extent. Our modern conceptions lead us to regard these functional tracts as nutritional entities and to term them neurons. Each depends for vitality on the cell from which the fibres proceed—a chief process ending in a

long fibre, and short processes quickly branching in the spongy grey matter. We have long recognised this relation to the cell, but the change in our conception has come, as you know, from the perception of discontinuity. The division of the fibres (which is really the separation of their constituent fibrillæ) terminates in ultimate branches which seem to end, but they end in contiguity to others from which they are stimulated. The groups of neurons which are differentiated in function, differ also in their tendency to decay—in their degree of vitality. When decay occurs slowly, it seems to be the result of a slow failure of the influence of the cell on which their vitality depends, and indications of degeneration are first seen in the parts that are furthest from the cell—in the extremities of the fibres. But these neurons differ not only in the lapse of life, but also in its early stage. Not only do they die at different times, but they vary in the time of their development. Some are structurally complete sooner than others, and thus are distinguishable in the beginning of life as well as in its ending.

Interstitial Overgrowth.

Before we consider the special forms of the slow failure of these neurons, the forms of neuronic abiotrophy, there is another feature of the process of decay which it is important to discern. Whenever the nerve elements waste there is always an overgrowth of the interstitial neuroglia, the connecting and supporting tissue which lies between them. This overgrowth may be, indeed, on first inspection, the most conspicuous element, and its aspect has led the process to be termed "sclerosis." I say "its aspect," because there is generally no increase of consistence in the part so changed. This is generally diminished; the interstitial tissue, which looks so fibrous and firm under the microscope, is really softer

than the nerve elements that have perished. This overgrowth has certain features which it is important to recognise. They have much significance in connection with conditions which we shall presently consider. The neuroglia is a residue of the embryonal tissue from which the nerve elements also develop. When there is local arrest of development of the latter, as in cases of congenital fissures and cavities in the spinal cord, this embryonal tissue persists in tracts adjacent. The two elements, the neural and neuroglial, seem to have a common but inverse vitality; it is in consequence of this that the interstitial tissue overgrows when the nerve elements decay. The overgrowth seems to coincide with the very commencement of the decay, and may be at first the more conspicuous. When it is thus started it often proceeds with an independent energy; in consequence of this, it may soon become much more obtrusive, and may overstep the strict limits of the degeneration and pass a little into adjacent tracts. It is especially luxuriant where the connective tissue is naturally most abundant, about the vessels and at the surface of the spinal cord, beneath the pia mater. On account of this the nature of the process was for a long time misunderstood. The significance of the extent of the degeneration, through the whole of a single tract of nerve fibres, did not receive its due weight as an indication that the degeneration of the nerve elements was the primary process. Attention was fixed upon the more conspicuous interstitial tissue, and the characters of this were thought to be evidence that it was the primary change and that it started from the vessels or from the membrane. The word "sclerosis" itself made the mistake easier, because there are some morbid processes in which this element *is* primary, as in disseminated sclerosis. For these reasons the degeneration of the motor tract in the lateral columns of the cord is still spoken of as "lateral sclerosis," and degeneration of the fibres in the posterior col-

umns is called "posterior sclerosis." I may add that we have another illustration of the tendency to independent growth of this tissue in the fact that the residual tracts which persist when the development of the cord is arrested, may take on a new process of growth at some period, usually in early adult life, so as to give rise to that which seems a definite new formation, the condition termed "gliomatosis."

Not only is the secondary overgrowth thus excessive in degree, but it sometimes assumes an energy which entails some vascular disturbance, as active tissue-growth is always apt to do. This may even be such as to constitute a process of an inflammatory character and it seems, even in rare cases, to be attended by hæmorrhage. At least there is occasionally a rapid or even sudden increase in the symptoms, not otherwise to be explained. Let me give you an instance. The optic nerve is a structure in which the amount of interstitial tissue which is formed in primary degeneration is exceptionally large. It is so in the simple atrophy of tabes. In this we sometimes have evidence that the interference with function is especially great at the optic commissure, so as to cause temporal hemianopia, an indication that focal damage is added to the primary atrophy. We can understand this if the secondary change assumes at the commissure an inflammatory degree. This is also indicated by the fact that such temporal hemianopia may develop rapidly. I have seen one case, and another has been recorded, in which, when such hemianopia had developed, it progressed to complete loss in one eye and then the remaining half field was suddenly lost, causing complete blindness, as if the vascular disturbance of the secondary process had led to a hæmorrhage at its chief seat. We also sometimes meet with evidence that axial retro-ocular neuritis, an interstitial process, develops in the course of simple atrophy, a fact which has the same significance. We shall presently see the importance of these considerations.

Special Forms.

Let us pass to the special forms of degeneration of the nervous system which seem to be abiotic. We may distinguish certain groups, to which I referred at the outset. The first consists of those in which the failure of vitality occurs before, or soon after adult life is attained, and it may be quite early, soon after structural development is complete. In many cases of this group the relation to an innate tendency is shown by their occurrence in several members of the same family, in brothers or sisters, and sometimes in more than one generation. But each form, even those in which a family grouping is most marked, may occur in isolation.

The natural transition from the muscular to the nervous system is by the fibres which end upon the muscles, the spinal motor neuron, whose vital centre is the cell of the anterior grey matter of the spinal cord. Note this: different as they are in aspect, of all structures the muscular fibres are nearest to the nerves. The muscular fibre is excited to instant contraction by the stimulus that reaches the extremity of the nerve fibre, which ends in contiguity to the muscle protoplasm, although the two are not continuous. So also the nerve impulse which stimulates the muscle arises, we now conceive, in the extremities of the branching processes of the cell, as the result of an impulse that reaches the ends of other fibres which are contiguous but not continuous. Remember, also, that the life of the muscle depends upon that of the nerve. The muscular tissue has indeed its own vitality, which may fade, as we have seen, independently; but it has a mysterious dependence on the nerves. Slow atrophy of the muscle follows the slow degeneration of the nerve. The atrophy might be thought to be due only to disuse, were it not for the change in electrical excitability which so soon presents itself—the "reaction of degeneration." But if the

degeneration of the nerve is of rapid, and not only of rapid, but of irritative character, the muscular fibres undergo a similar change, a more irritative degeneration, a quick destruction, with granular and fatty degeneration and a rapid disappearance of all electrical excitability. These facts indicate a close vital relation between the muscular and nervous structures—a relation we may call one of functional vitality, as distinguished from inherent vitality.

Of the many forms of abiotrophy in the nervous system, perhaps the most striking is one of which these spinal motor neurons are the seat. It is rare but it has been observed many times.* It commences soon after they have attained structural completion, about the end of the first year of life, and progresses so rapidly as to cause almost universal paralysis and death at the end of the fifth or sixth year. It occurs in families as well as in isolated cases, and also in both sexes, and thus must be ascribed to a congenital defect of vital endurance. After death there is found almost complete degeneration of the motor cells of the spinal cord, with very little change in the white columns, degeneration of the anterior roots and of the motor fibres down to the muscles, and extreme degeneration of these. It has, indeed, been conjectured that both the motor neurons and the muscles suffer simultaneously; whether it is so or not we cannot say, for the affection of the motor neurons is sufficient to account for the muscular changes. It is quite certain that the degeneration of the motor nerves is not a consequence of the muscular atrophy. In extreme idiopathic muscular atrophy the nerves may be unaffected. A striking proof of this is afforded by a case which was observed by Duchenne and

* See especially Hoffmann, "Deutsche Zeitschrift für Nervenheilkunde," Band 18, 1900; Ibid., vol. xx. Cramer, "Centralblatt für Allegemeine Pathologie," 1895. Senator, "Charité-Annalen," xxvi. "Jahrgang," &c.

published by him when the patient was nine years old; it was afterwards observed and described when the patient was 28 years old by Landouzy and Déjèrine. The patient died when she was 36 years old, but the thorough pathological examination that was made has only lately been published.* In spite of extreme degeneration of a large number of muscles of limbs, trunk, face, and neck, the motor-nerves and spinal motor-cells were absolutely normal.

But the abiotic infantile atrophy presents one remarkable feature; the affection begins in the muscles of the hip and thigh and trunk, and thence extends, reaching last the extremities of the limbs, which suffer first in the ordinary form of spinal atrophy. This is a feature presented both by muscular abiotrophy and by this remarkable neuronic form. Such common features are noteworthy as links between the muscular and nervous systems. A similar form of neuronic atrophy is occasionally met with in later childhood and early adult life. In some forms the cerebral motor neuron also suffers, the upper segment of the motor tract, as it does in the atrophies of later life. The isolated decay of this cerebral motor neuron is also met with in early adult life as an abiotic degeneration of the lateral columns—the pyramidal tracts. It is manifested by spastic paraplegia, coming on often without any exciting influence and sometimes in several members of the same family.† Occasionally the malady has followed some influence, such as an acute disease, which would depress the nutritional vigour of the whole nervous system, but has an enduring effect only on structures whose vitality is defective, and might soon fail without this influence.

* Spiller, Cont. Pepper Labor., Philadelphia, 1900.

† See, for instance, the cases recorded by Dr. Howard H. Tooth, "St. Bartholomew's Hospital Reports," vol. xxv.; Strumpell, "Neurologisches Centralblatt," 1901, p. 628; Krafft-Ebing, "Deutsche Zeitschrift für Neurologie," 1900; and Finzi, "Rivista Sper. di Fren.," xxi.

I have here to show you an example of such abiotic spastic paraplegia. It is the more instructive because it is isolated; such sporadic cases are far from rare. Their nature is often not discerned, because it is not suggested as it is when other members of the family suffer. The patient is 20 years of age, and his symptoms commenced gradually about three years ago without any cause to which they can be attributed. There is no suspicion of inherited or acquired specific disease. He has weakness of the legs, with some spasm, excessive knee-jerk and foot-clonus, with no change in sensibility, and no girdle pain. He has also some unsteadiness, suggestive of some failure in the posterior columns. The abiotic nature of the case is confirmed by the fact that another system of fibres is also failing. He has atrophy of the optic nerves which also has developed gradually and presents the aspect of simple atrophy. The simultaneous occurrence of such degeneration in two neuronic systems at this period of life leaves no doubt of its nature.

Optic Abiotrophy.

The optic nerves frequently suffer from abiotic wasting. Many groups of cases are on record in which several members of the same family have become blind, usually between the ages of 15 and 25 years, from a slow failure of the nerve fibres, progressive in spite of treatment. A full account of these cases has been given by Dr. S. H. Habershon.* This progressive course places their essential nature beyond doubt; yet it is obscured by the fact that the early stage has very often been accompanied by symptoms of retro-ocular neuritis, even with slight signs of it in the disc. There has often been

* "Transactions of the Ophthalmological Society of the United Kingdom," 1888, p. 190, where references to the chief writings on the subject will be found.

a central scotoma indicative of an "axial neuritis." The change, moreover, has often been preceded by a cause capable of inducing such neuritis, especially tobacco-smoking, or in the case of women some acute disease. We may understand this fact better, I think, if we remember what I said just now regarding the independent energy of the interstitial overgrowth that concurs with degeneration of the nerve elements. We may also remember the great amount of interstitial tissue that is formed in the optic nerves, even in primary tabetic atrophy. Tobacco amblyopia is apparently due to an axial neuritis, which gradually ceases, and its effects pass away when the cause ceases to act. But the loss of sight, in the cases I am now speaking of, goes on steadily increasing after tobacco has been given up. It goes on as by a progressive atrophy of the nerve elements, without any increase of the signs of neuritis, showing that the toxic agent was only the exciting cause of the process really due to an inherent defect of vitality. In those who are not smokers the toxine of an acute disease seems sometimes also to act as an excitant. We know that the toxine of scarlet fever may cause pronounced optic neuritis. The difficulty is to understand that optic abiotrophy, wasting of the nerve-fibres from defect of vital endurance, should apparently begin by interstitial inflammation. But let me remind you of what I have just said regarding the interstitial overgrowth which attends the atrophy of the nerve elements. Secondary as it seems, it may be simply simultaneous. There seems to be a solidarity in the nutritional energy of the two tissue elements, the neural and neuroglial, and the tendency to overgrowth of the latter may be synchronous with the tendency of the life of the former to cease. The tendencies are in the opposite direction, but they seem to be coincident results of the same vital condition. If so, we can understand that the tendency of the interstitial tissue may cause it to respond with readiness and

energy to a toxic influence which can act upon it, and that thus retro-ocular neuritis, the effect of a toxic agent, may concur with the very commencement of the abiotic wasting of the nerve elements, and that its special symptoms may even *seem* to lead the way. It is not a little curious that in the patient before you, with abiotic spastic paraplegia and optic atrophy, we find indications of the same associated process that I have described. As far as its course and the ophthal-

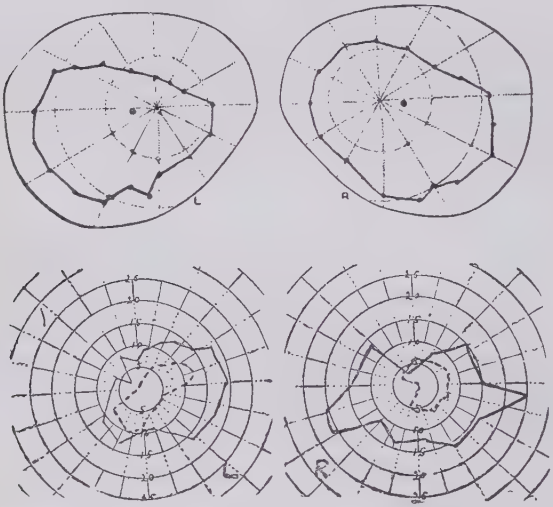


Fig. 14.—Fields of vision. The upper fields are for white; the lower represent the central region, the field for red (continuous line) and green (broken line). In the upper figures the outer line is the normal field.

moscopic appearances are concerned it seems to be a primary simple atrophy of the optic nerve. His acuity of vision is reduced to one-tenth. The fields present a peripheral restriction, but no central defect in the fields for white can be detected, even with a small object. Yet it is different with the colour fields. There is a well-marked central scotoma for both red and green (see accompanying chart). It is

irregular in outline, but extends to an average of about 10° from the fixing-point, and in one eye to 20°. This, of course, suggests axial neuritis, and led to an inquiry regarding tobacco-smoking; we learned that he had been accustomed to smoke as much as half an ounce of shag tobacco a day! Thus this agrees with other cases of optic abiotrophy which I have mentioned in presenting slight symptoms of axial neuritis and in the fact of the same exciting cause. But it presents the progressive tendency and also the peripheral restriction of the field and the aspect of disc met with in ordinary atrophy, while its abiotic nature is indicated also by the coincident affection of the cerebro-spinal motor neurons.

Friedreich's Disease.

The spinal symptoms in this patient, the spastic palsy and unsteadiness, due to atrophy of the motor neurons and those which conduct to the cerebellum, are such as often form part of the best known abiotrophy, Friedreich's disease, the so-called "hereditary ataxy." The latter name is another instance of inaccurate terminology, because the evidence of heredity is not so common as is the affection of collaterals, of brothers and sisters. This "family" feature is clear proof of the congenital nature of the tendency to vital failure which the disease manifests. Friedreich's disease is really ataxic paraplegia, depending upon slow changes in the posterior and lateral columns, but associated with other symptoms in speech, etc., and in it the abiotic tendency is clearly congenital. Males suffer more frequently than do females, but not in the same degree as in some other analogous maladies.

Friedreich's disease was the first of the kind to be distinguished and is the best known, but observation has revealed the wide variation in the precise characters presented by cases which cannot well be separated from typical forms.

The characteristic symptoms, the weakness and unsteadiness of the legs, sometimes ataxy of the arms, the change in speech, and the nystagmus, are often found to be associated with some impairment of sensation, and with either loss or excess of the knee-jerk; the condition of speech also is found to vary, and there may or may not be mental alteration. The cases pass by degrees into another group which has been thought to be distinct. In this the most conspicuous feature is the swaying disturbance of equilibrium when standing, with or without evident weakness of the legs; there may be loss or excess of the knee jerk, but there is seldom any disturbance of cutaneous sensibility. The arms may present any form of incoördination or none. Speech is commonly altered, slow and monotonous, sometimes with confluent articulation, sometimes with a staccato utterance. There is almost always nystagmus, and mental change is more often marked than in Friedreich's disease. The difference in other symptoms is generally one of degree only and depends on the age at which symptoms come on. This is chiefly during the second half of actual childhood. Resemblance of the symptoms to those of disease of the cerebellum has caused the malady to be called by the inexact and clumsy name "heredo-cerebellar ataxy." Although heredity has been found in a few cases, merely a family association has been commonly met with. Disease in the lateral columns has been found, but the changes are greatest in the posterior columns and the direct cerebellar tract, which seem to convey afferent impulses to the cerebellum; their defect deranges its coördination of impulses that guide the cerebral motor centres. Damage to these upbearing fibres, conveying impressions from the muscles, seems thus to cause this peculiar "cerebellar" ataxy, as distinguished from the pure incoördination of the legs met with when the inbearing fibres to the cord degenerate, as in ordinary tabes. All the facts regarding the disease show that it is a true abi-

otrophy, a degeneration of certain neurons from defect of vital power, but the precise distribution of the defect varies in different cases. Probably many of the neurons in the brain which connect different parts, which associate and harmonise their functions, also waste, and thus the mental change may be explained. But the special pathology of this affection is beyond my present subject.

Before I leave this group of abiotrophies, those of earlier life, it may be well to point out that not all the morbid states which seem congenital are really such. Caution is especially needful in the case of the diplegias which date from birth, because they are so frequently due to meningeal hæmorrhage during a tedious or instrumental labour. The narrow pelvis, which causes this in one child, may have the same effect on another, and thus we may have the semblance of a family affection in that which is purely acquired. The birth damage tends to improve as time goes on; abiotrophies tend to increase, although when slight in degree they seem sometimes to become stationary. The varying course observed in both muscular and nerve affections makes it probable that the degree and extent of the deficient vital endurance may vary much in different cases, with corresponding variations in the tendency of the symptoms that it causes.

Later Neural Abiotrophy.

The second group of degenerations are those which occur at the other end of life. While general vitality still seems full of vigour, the nutrition of some neurons fails; they slowly die. The neurons which most frequently thus decay are the spinal motor neurons—those which sometimes fail, as we have seen, at the very beginning of life. According to the position of the tracts which suffer first and most, the effect is the symptoms of spinal progressive muscular atrophy or of labio-

glossal paralysis. These are more frequent in late life than isolated degeneration of the upper motor neuron which causes spastic paralysis, although this sometimes suffers alone, and almost always degenerates when the lower neuron suffers. These maladies often come on without any apparent cause, simply because the term of life for those structures is reached sooner than for the vital organs; they decay in a true abiotrophy. When any exciting cause can be traced it is usually one which is inadequate alone—some general depressing influence, such as anxiety or grief, which must act equally on all the other structures and tissues, reducing the vital energy of all. But vitality only fails in those which have inherently a less degree; their defect is sufficient to make the reduction in these, though the same as in the others, relatively greater in effect, sufficient to end their life. Without this influence they might or might not have persisted to the end of general life. The apparent cause does not lessen the significance of the facts which show that these affections are essentially senile abiotrophy.

Mental change, especially simple mental failure, often occurs under the same conditions, and no doubt from a slow degeneration of cerebral neurons which connect and combine others in a way we cannot yet perceive. Another senile malady, paralysis agitans, must be referred to vital failure in some cerebral motor structures. In its conditions and course, it is a striking example of a cerebral abiotrophy. In spite of its obvious seat in motor structures, in spite of its strongly progressive character, it seems never to entail the decay of the motor neurons which connect the brain, the spinal cord, and the muscles. It formerly seemed to me like a degeneration peculiar in kind, such as we are reminded of by that which causes the hair to become white from lack of pigment though growth is still luxuriant, which presents a contrast to the degeneration which causes baldness. But the recent discov-

eries compel us to look to the extremities of the branching processes of the motor cells, the dendrites which pass into the spongy grey substance, for the source of the motor impulses which are conducted by the fibrils that pass through the nerve-cells without interruption. A change in the nutrition of the dendrites may disorder function, yet leave unaffected the vital cell and the nutrition of the neuron as a whole. It is also conceivable that there may be a change in the relation of the dendrite endings to those which stimulate them and induce the motor impulses. We know too little to be able to form an adequate hypothesis. But the conditions under which paralysis agitans generally occurs and its course show that it must be regarded as the expression of a special defect in vitality—as a form of abiosis. Its occasional occurrence, even in extreme form, in middle life, also without discernible cause, is consistent with this view, for the same is true of other senile maladies.

Toxic and Toxinic Degenerations.

A third group of degenerations remains, large and important. They are the varied degenerations that occur, especially in middle life, as the result of some definite cause. It is not clear how far these are due to a defect of life. The degeneration may be the result of the presence in the blood of some material which is able to enter into the constitution of the nerve elements, but is not adequate for their proper functional or structural maintenance. They may slowly recover if the supply of the noxious matter ceases. Arsenical neuritis is an example. Arsenic seems capable of taking the place of phosphorus in metabolic changes of nutrition, but its presence deranges function and also structure, so that degenerative change is the result. The alteration begins where vitality is weakest, at the extremities of the nerves furthest from the

nutritional centre, the cell; the energy of this may ultimately be impaired, but we have not evidence that the primary influence is exerted on the cell or that the process consists essentially in a failure of vital energy—that it is a true abiotrophy. The same statements may be made with regard to alcoholic neuritis, since the process in this has the same features.

The most common neuronic degeneration of the middle period of life is tabes. This, as you know, must be ascribed to the influence of a post-syphilitic toxin. But it occurs so long after the primary disease and at such a variable interval of years, that the question arises—Is the affection of the nerve elements due to the constant presence of the toxin in the system, in varying quantity and varying degree of toxic power, or has a transient toxin an influence in reducing future vital endurance? In the latter case it may be regarded as acquired abiotrophy. Each hypothesis has difficulties, but neither is quite inconceivable. On the whole, I think the first is that which best explains the facts of the disease. The analogous toxin of which we know most—that which causes diphtheritic paralysis—has been proved by Sidney Martin to be due to a change in the chemical constitution of albuminoses, caused by a ferment-like material which the organisms in the throat produce. The toxin varies in its character, to judge by its different effects, and it is noteworthy that it may sometimes act on the same neurons as are affected in locomotor ataxy and give rise to the same symptoms. It is conceivable that such a change in the chemical constitution of albuminous substances, once set up, may continue, and the toxin may be constantly produced, in varying amount and varying toxic power, its formation being modified from time to time by other influences of which we know nothing. In the processes concerned in the production of excess of sugar, we have an example of the persistence of the derangement of

a chemical process, and the gradual increase of the derangement if not restrained, and although the analogy is not very close (since sugar is a normal constituent of the blood) it illustrates that such disordered chemistry may be enduring. The occasional rapid increase in the symptoms of tabes, such as may bring a patient from a stationary slight degree to the most extreme disability in a few days, is better explained by the sudden production of a poison of greater amount or power, than by the sudden reduction of the resistance of structures the vitality of which is impaired. The same conclusion seems suggested by the great improvement which we meet with in some cases. A man came in here about two years ago who had had, for some years, symptoms of the first stage of tabes, and then in the course of a few days the ataxy increased to such a degree that he was not able to stand, from the extreme incoördination. Retention of urine had developed and cystitis had set up a suppurative orchitis. Yet this patient gradually improved until he could walk without much unsteadiness before he left the hospital, and now he can walk well for eight or nine miles. Unfortunately, improvement does not always occur in such cases of rapid exacerbation. There is one tabetic process the course of which is more uniformly progressive, so much so, indeed, as to resemble closely the effect of reduced vitality—the atrophy of the optic nerves. But even in this we meet with cases which become arrested, usually as the result of treatment. The significance of this fact we cannot discern or even wisely speculate about.

Arrest or improvement from treatment occurs in other slow degenerations which occur during the middle period of life; some of them are met with occasionally as the sequelæ of syphilis, but they may occur without obvious cause or as the result of exposure to cold, or they may seem to be due to the toxin of some other specific disease. The

nature of these also is doubtful. Some of the progressive muscular atrophies of middle life resemble the senile form we have just considered in their slow commencement and gradual extension and increase, and often in the absence of a cause. Yet they differ in being more amenable to treatment. There is a considerable prospect that such cases will be permanently arrested. But even in such cases after syphilis, it is not by anti-syphilitic treatment that good is done, but by the hypodermic injection of strychnia. In one typical case the malady commenced just after a course of treatment at Aix, but the permanence of the arrest induced by strychnia is shown by the fact that it has now been absolute for about twelve years. In another case, more advanced but also arrested, the treatment was commenced after a rapid increase which occurred during the administration of iodide of potassium. This treatment has not the same influence in the senile abiotrophic form.

That terrible malady, general paralysis of the insane, seems, often at least, to be due to the same cause as tabes, symptoms of which are so often associated, and seems to be of the same degenerative nature, produced no doubt in a like way. That which has been said of tabes applies to it also, and that which has been said of the interstitial alterations, and their independent energy, may explain the inflammatory changes often met with. But here we are on uncertain ground. The pathological relation of the morbid process to its cause is quite obscure.

Just as the simple baldness which develops in early manhood may also come on at any period in adult life and in any degree, so the family abiotrophies which usually commence early in adult life or in childhood may show themselves later. In the families in which some members suffer early, in other members the same symptoms may occur only during middle life—a fact which we can understand by the concep-

tion that the degree of vital energy in such cases is greater, the vital endurance is such as to persist longer. Yet the occurrence of both early and late forms in the same family shows that their nature is the same and that the difference is one of degree.

Conclusion.

These conceptions I hope may help you, if not to understand these processes, at least to think of them more clearly. I would especially impress upon you the fact which is illustrated by the lad whom I have shown you—the sporadic occurrence of these abiotrophic diseases. Their nature is easily discerned when they occur in groups which have the tie of consanguinity, but the discernment is less easy when the diseases occur in isolated form. Remember the significance in such cases of the age of the patient, the slow development of the malady, the absence of adequate cause, whether an inadequate cause can or cannot be traced, and the correspondence of the symptoms to the functions of certain sets of neurons. Many cases of this kind are regarded as due to disseminated insular sclerosis, often assumed to exist when the symptoms afford no justification for the opinion and in cases in which the assumption is disproved by the future course of the disease. Remember that the symptoms of systemic degeneration are not alone ground for the diagnosis of insular sclerosis, nor is the additional presence of nystagmus. Nystagmus on movement of the eyes is met with in many spinal degenerations of pure systemic type, why we do not know, but the fact deprives it of the diagnostic importance which has often been attached to it.

My object in this lecture has been to present to you the fact that many degenerative diseases of the nervous system are a result of a defect in vitality. The two aspects of death which I mentioned at the outset are very different in our

practical view. Somatic death is that against which we ever strive, which we endeavor to postpone as long as may be. The other form of death—the termination of life of isolated structures in the body—is to us what we call disease. It does not of necessity involve the termination of general life, but it is not less to be striven against, although the strife must often be in vain. The discernment of these diseases, which we may call "abiotic," and the recognition of the symptoms, the course, and the conditions which indicate them, is of extreme importance. Their discernment may save you from many errors and may prevent many mistakes, both in your forecast and your treatment. We must endeavour to check their progress, for we seldom know the strength or feebleness of the tendency, or whether it can or cannot be hindered, but the perception of the nature of these maladies will often help you. It will enable you better to perceive why treatment fails, and it may save from useless prolongation of attempts to gain that which cannot be, and it may sometimes save from waste of money that can ill be spared, and from the waste of hope which means only deeper disappointment.

LECTURE IV.

MYOPATHY AND A DISTAL FORM.

Delivered at the National Hospital for the Paralysed and Epileptic.

Gentlemen:—In the last lecture I gave here, I considered the various maladies that depend on defective vitality, on the imperfect vital endurance of the tissues which causes their nutrition to fail, generally early in life, sometimes later. I suggested that this decay, which results from defect of life, might be called "abiotrophy," and the word has been welcomed as convenient and useful. I illustrated the condition, as some of you may remember, by the wasting of the hair follicles of the skin which causes premature baldness, by the forms of muscular dystrophy which often occur in families, and have thus their essential vital nature strongly emphasized, and also by the many forms of degeneration in the nervous system that are of the same nature, which vary according to the special structure that fails, the time of life at which the vital failure begins, and the cause which sometimes seems to be its incidental excitant.

I have to return to the same subject to-day in one of its special manifestations. We are all opportunists, more or less, and I am able to show you some examples of muscular abiotrophy which are so instructive as to compel me to take them as my subject to-day. You know that these primary affections of the muscles are called "idiopathic muscular atrophy," "myopathy," and also "muscular dystrophy."

* British Medical Journal, July 12, 1902.

The last name (due to Erb) means "difficult nutrition," and has a certain aptness, because there is often an overgrowth of the interstitial tissue which suggests the idea of a struggle for existence, such as we sometimes see between weeds and the proper crop which has been sown on the ground. The metaphor is not quite precise, nevertheless we shall see that it is not without a deeper justification. In the form which is termed "pseudo-hypertrophic paralysis," of which I am fortunately able to show you two instructive cases, this interstitial growth attains a special luxuriance. Fat cells are formed in the fibrous tissue that develops, and from this there is an enlargement of some muscles, whence has come the common name of the disease. It was given by Duchenne, who had been specially struck by this feature, the more so because it had chanced to him to meet with some cases in which the enlargement attained a rare extension and degree, being present in most of the muscles. He used to claim Raphael's child-angels as examples of the disease. Some of you may remember the extraordinary development of their muscles depicted in the celebrated cartoons. In the early days of this Hospital there hung, over the fireplace in the consulting-room, an engraving of Raphael's picture, "The Transfiguration"; in it is shown a child with demoniacal possession; it was placed there as an appropriate representation of an epileptic fit, or the most accurate known in art. On a visit Duchenne once paid to the Hospital, I remember that he pointed out the figure as one which might well pass for an example of the disease on which he had bestowed the name.

But there may be no increase in size of the muscles, or there may be a decrease. The interstitial tissue may merely compensate for the wasting of the muscular fibres or it may fail to do so. Hence, in the same case, we meet with muscles that are weak and large, that are weak and normal in size,

and that are weak and small. Interstitial growth, which is luxuriant, or moderate, or slight, is seen in the same case; one or the other may preponderate in different cases. Names are often instructive, but they sometimes obscure. I think that Duchenne's designation hindered the recognition of the fact which has been established by Erb and others that neither the amount of interstitial tissue, nor its character, is an essential element of the disease. The constant element is the change in the muscular fibres themselves; some of them may appear at first unduly large, but many are small and present the features of degeneration; ultimately this becomes general, irrespective of the increase of the interstitial tissue. It is a true abiotrophy.

As to these differences, let me remind you of what I said in the last lecture regarding the growth of interstitial tissue which occurs in the nervous system when there is primary atrophy of the nerve elements. I pointed out the variation it presents, and that it sometimes attains a luxuriance and energy which make it overstep the strict limits of the degenerated tracts. This feature at one time raised a suspicion that it was the primary element in the process, and a similar suspicion regarding the muscles was excited at first by the abundant development of interstitial tissue, especially in the pseudo-hypertrophic form. Let me remind you also of another fact I pointed out. When we consider the features of this double process, the atrophy of the functional structures, and the overgrowth of the interstitial tissue, they suggest that the two elements have a vital relation, common in the necessary connection of the life of each, but opposite in tendency. When the nutrition of the muscular or nerve fibres begins to fail, that of the interstitial tissue is increased. It does not grow merely to fill up space; it is not truly a secondary process, it is the expression of an excess of vitality simultaneous with the diminution of that in the proper

structures. The two tendencies, plus and minus, seem to be synchronous in these cases of vital failure, and the excess may have results at first more conspicuous than the diminution. The interstitial growth may be at first much more obtrusive than the atrophy of the functional elements. We saw how, in the optic nerve, it seems to promote the response of the interstitial tissue to the influence of a toxic agent (tobacco) in the form of interstitial neuritis, the symptoms of which lead the way, but at an earlier age than the toxic influence alone is effective. Moreover, when this influence ceases to act, the usual improvement does not occur—the abiotrophy still goes on. The same initial excess of vitality of the connective tissue seems to induce the exuberant development of the interstitial tissue in the muscles, and the production of fat cells within it, in muscular abiotrophy.

The degree of overgrowth varies in degree in different parts and also in different cases, from causes we cannot discern, yet we can understand its features better if we remember the double character of the process. But the excess of the interstitial vitality is only temporary. It often attains a conspicuous degree before the failure of the life of the muscular fibres is considerable, but afterwards, as their failure becomes great, the vital energy of the interstitial tissue fails also. It is an initial outburst of energy; ultimately it also shares the vital failure. The fat becomes absorbed, and the fibrous tissue gradually contracts, so that the muscles, at first large, lessen but become more firm, and those which are not enlarged shrink; ultimately almost all become small as they become more and more feeble, only some trace of enlargement may linger long where it was at first so great.

We shall see these states in the patients I have to show you. They determine variations in the aspect of cases in which the same muscles suffer in different ways, and still

greater variations are due, as we shall see, to the difference in the distribution of the affection. Under the microscope are some sections which are worth your inspection. One is from the deltoid muscle of a case of pseudo-hypertrophic paralysis, and in it you can see the excess of fibrous tissue and the fat cells between the muscular fibres, and that many of these are narrowed and others are undergoing structural degeneration. Under another is a section of the lumbar enlargement of the spinal cord of the same case; in this you will see that the grey matter and nerve cells present a normal aspect, although the condition of the legs was one of almost complete loss of power. Under the third microscope I have placed a section of much interest as well as rarity. The part of the section under the microscope looks precisely like a section of muscle in pseudo-hypertrophy. Narrow striated muscular fibres course among tissue composed mainly of fat cells with some strands of connective tissue. But it is a section of a small tumour attached to the spinal cord—a myolipoma. It had caused no symptoms, and was discovered by accident, at least if the phrase can be applied to that which is revealed by a careful search, without definite object. But it is instructive, because it is evidently a fragment of the embryonal tissue of muscle which is developed in a wrong place, and in a wrong way. It shows how morbid development results in the condition we find in this disease, and illustrates its truly "abiotrophic" nature.

Pseudo-hypertrophic Form.

I cannot, for lack of time, and I hope I need not, describe the general symptoms of pseudo-hypertrophic paralysis, but I may point out some symptoms that are illustrated by the cases I have here. The first is a boy of seven, who is still in the early stage. His case seems alone in his family. I in-

sisted in the last lecture on the fact that all family maladies are prone to occur in isolated form, and how important it is to learn to recognize them by their characters and course, because so often you have not the evidence of their nature afforded by other cases in the same family.

In this child no muscles are sufficiently increased in size to attract much attention, but the calves are distinctly large and firm. The knee-jerks are slight, but are not lost, as they are in the later stage of the disease. He is just acquiring the characteristic method of getting up from the floor, aiding the extension of the knees by putting his hands on them. This transfers some of the weight of the trunk from the upper end of the femur to its lower end, from the place at which the power exerted by the rectus acts to least advantage, to the place near the fulcrum at which the advantage is greatest. You may ask if this action is due to the weakness of the extensor of the knee, why is it characteristic of this disease, since these muscles are often weak from other causes? The question is a reasonable one. The action is not absolutely characteristic; it is met with in other maladies, but in such it is very rare. This particular method of aiding the defective movement seems to be acquired only when the weakness comes on slowly during the development of muscular ability. As a matter of fact it is so rare under other conditions that its diagnostic importance is very great.

The boy presents two other symptoms, the significance of which I was, I think, the first to point out.* One is that the infraspinati are enlarged quite as conspicuously as the calf muscles. When the muscle is put in action, as you can see, it appears as a rounded prominence. The other fact is the atrophy of the latissimus dorsi and lower part of the pecto-

* "Pseudo-hypertrophic Muscular Paralysis." London: Churchill. 1879.

ralis major. These sometimes are absent, and often, as here, are reduced to small dimensions. Their loss is often so great, even at an early stage, as to suggest a congenital defect.

The disease is far advanced in the second boy I show you. His weakness became conspicuous at the age of seven, and he is now fourteen. He lost the power of standing four years ago, about a month after an attack of scarlet fever. The acute disease seemed to induce a rapid increase in the symptoms; you should note this, because the symptoms of abiotic failure often first attract attention after some acute disease, such as scarlet fever or influenza; and the malady is ascribed to this influence, although it really existed before. It is easy to understand that an affection commencing gradually may have been unnoticed, and that a depressing influence, which really only accelerates, may seem to be the cause. In this boy the calves are not absolutely large, but they are so in comparison with the other muscles, which are extensively wasted, and are weak even out of proportion to the wasting. He has hardly any power of flexing the ankle or extending the knee or hip, but these movements are also prevented by the great contraction which has supervened in the opponents. Permanent shortening occurs very readily in this disease from contraction in the interstitial tissue. When the power of standing has been lost, such shortening quickly fixes the limbs in the position naturally assumed in the sitting posture. In this child the contraction of the calves is great, and that of the flexors of the knees and hips is such as to prevent extension beyond a right angle. The calf muscles contract earliest and most, doubtless on account of the amount of interstitial tissue in them. The talipes equinus that results, often prevents the patient from standing and walking, long before the weakness of the other muscles would do so. Once the muscles lose the nutritional influence of

use, they rapidly become weaker, and this weakness, once established, is irremediable. We shall presently see the practical importance of this.

We can still perceive some enlargement of the infraspinati, although less than in the former case; the characteristic atrophy of the latissimus dorsi and lower part of pectoralis is present in an extreme degree. But of all the muscles of his body, that which is enlarged in greatest degree is the lower part of each serratus magnus. You can see how these muscles stand out when he endeavours to push his arms forward. I have seen it before, and indeed it is shown in a figure in my "Manual," but it is not common. The influences which determine the character of the morbid process in the different muscles are quite unknown to us.

His arms present characteristic weakness, least in the forearms and hands. The muscles of the neck are so feeble that he has some difficulty in holding up his head. The clavicular part of each sterno-mastoid is hardly to be detected, and this is a noteworthy feature, common to many forms of myopathy.* The muscles of the face have here fair power for the most part, but the closure of the eyelids is feeble, and the left orbicularis is much weaker than the right. This is important; the difference between the two sides emphasizes the morbid character of the weakness. I may add that the electrical excitability of the muscles is lessened in both of these patients in proportion to the wasting, equally to each current; this is the characteristic condition in these diseases. In this patient the knee-jerks can no longer be obtained. Their gradual disappearance is an almost constant feature.

*I noted this feature as long ago as 1879. ("Pseudo-hypertrophic Paralysis.")

Facio-scapular Form.

The weakness of the orbicularis palpebrarum, which I have just pointed out, is of interest as another of the many links which connect the various forms of muscular dystrophy. It is especially great in a variety in which the face is affected in extreme degree, and so are the muscles of the shoulder and upper arm and trunk. As a rule, in this form, wasting accompanies the progressive weakness without conspicuous enlargement. From the distribution it has been called the "facio-scapulo-humeral type," and also, from those who called special attention to it, it has been designated the "type of Landouzy-Déjerine."

I show you now a striking example of this form, and his aspect is strangely similar to that of a patient of whom figures are given in my "Manual."* I could fancy this was the same patient a few years older. I am able to show him to you by the kindness of my colleague, Dr. Beevor, under whose care the patient is. I must leave it to him to demonstrate the special features, but I would ask you to note the extreme weakness of the orbiculares palpebrarum. It is so great that the patient cannot completely close the eyelids. Common as is the palpebral weakness, in few diseases of this class does it attain the degree seen in this form of myopathy. Yet a characteristic case of myasthenia has lately come under my notice in which the eyelids cannot be brought together by any effort—another link between myasthenia and myopathy.†

Extreme as is the weakness of many of the muscles in this facio-scapular form, great as is the ultimate degree of their atrophy, however long its duration, the malady seems to be purely muscular; the nervous system, even the motor nerves,

* Vol. I, third ed., Figs. 169–171.

† British Medical Journal, May 24, 1902.

are unchanged. The fact rests on many observations, on no one more striking than that which has been recently published by Spiller of a case described in the early seventies by Duchenne himself, in which life only terminated at thirty-eight.* We might wonder that disuse did not involve structural change in the course of so many years; but we must remember that the inability of the muscular tissue to respond to the motor impulse does not mean that the motor impulses themselves are less than normal. This question, however, would lead me far from my present subject.

Distal Myopathy.

The last case I have to show is one to which I would specially direct your attention because it presents unusual features. Indeed, I have not seen a similar case, nor do I know that one has been recorded, but it is always improbable that any given morbid state has not been seen before, and has not been described somewhere in the vast expanse of medical literature.

This boy is eighteen years of age, and is the eldest of three; the others are healthy. No similar case can be heard of in his family. The symptoms first attracted notice when he was ten or twelve years old; then it was noticed that he often caught his toes against the ground in walking. At a later date his hands were found to be weak. This feebleness of hands and feet has slowly, steadily increased, until now it is great. He is quite unable to flex the ankles, although he can just extend the toes, and can move each foot slightly in and out by the tibialis anticus and the peronei. He can extend the ankle-joint by the calf muscles with some force. The movements of the knee and hip are performed with good

* Proc. Pepper Lab., Philadelphia, 1900.

power; the knee-jerks are present, but the left is less than the right. The thigh muscles are of normal size; the anterior tibial muscles are distinctly smaller on the left side; the calves, on the other hand, are rather large, and are firm. They are sufficiently large to suggest a resemblance to the condition in pseudo-hypertrophic paralysis, and they may have been larger in the past, for his mother spontaneously told me that their size had often attracted attention.

Similar weakness is present in the upper limbs. His grasp is extremely feeble. With the right hand he cannot move the dynamometer, and with the left he can only move the index to 2 K°, instead of 50 or 60, as he should. Extension is also feeble; he can get the fingers, with the wrist, into line with the forearm, but cannot fully extend them when the wrist is over-extended. This is a convenient index of slight deficiency of power in these muscles. The muscles of the forearms and hands are small, but present no wasting comparable to that which we see in progressive spinal atrophy. Above the forearm, the muscles have fair power and present no wasting; only a trifling atrophy of the middle part of the trapezii can be observed. In the affected muscles electrical excitability is lowered in proportion to their feebleness, and equally to faradism and voltaism.

But this is not all. His neck muscles are normal, with one noteworthy exception—the sterno-mastoids. Of these only a trace remains—only a small fasciculus of the sternal part can be put in action by the will or electricity. The clavicular part seems quite absent. The condition is of such a character, and is so similar on the two sides, as to suggest rather a congenital defect than an actual atrophy. It is a feature connecting the condition with other myopathies. We have just seen a similar deficiency in pseudo-hypertrophic paralysis. The platysma here seems also absent.

The condition of his face is noteworthy. The movement

of the lips seems unimpaired. His tongue, however, presents a curious aspect of wasting on each side, yet it moves properly, and there is no impairment of the palate. His smile, especially on the right side, presents too little movement outside the corner of the mouth in proportion to the elevation of the upper lip. But the most important weakness is in the upper part of the face. He cannot raise the eyebrows at all; the frontales are powerless. Closure of the eyes by the orbiculares is also weaker than normal; their contraction is easily overcome, and more easily on the left side than on the right, conclusive evidence that it is morbid. This weakness of the orbiculares is met with, as I have said, in many forms of myopathy; it is thus of considerable significance, and so are the weakness of the frontales, and the atrophy of the sterno-mastoids. All these features stamp this case as a primary myopathy, and yet it differs from all recognized forms in the purely distal distribution of the affection in the limbs, and the normal state of the muscles near the trunk. In other forms the preponderant weakness and wasting are in the muscles moving the proximal parts, of the shoulder girdle and the hip, of the elbow and the knee. In the upper limb this is very conspicuous. In the leg the affection of the calf muscles is usually attended with weakness of the flexors of the ankle, but even in the early stage the loss of power in the thigh muscles and those of the hip is greater, and as the disease advances the preponderance of their atrophy becomes more and more manifest. Yet in this lad these muscles have good power, while any flexion of the ankle-joint is impossible, not from contraction of the calves but simple failure of the flexors. The feet can be moved inwards and outwards, and the movement outwards by the peronei is a distinction from the "peroneal form" of muscular atrophy, which, moreover, is believed not to be a true myopathy. Peculiar as is the distribution of the weakness, the

symptoms in the face and neck associate it with other forms and compel us to regard it as of the same nature, an example of what we may term "distal myopathy."

It is indeed remarkable how many symptoms, small we may regard them, link together the various forms of myopathy, and how important therefore is their recognition. Not less remarkable is the way in which the general type is maintained in families in which several cases occur, and hardly less important are the variations from the type which individual cases frequently present. These variations leave the general character of the malady clear, and yet they connect one type with another. I wish I could show you another case, which affords a pertinent illustration of this connection —so pertinent indeed that I must tell you the chief features. The distal form, with weakness of the hand only, which I have just shown you, presents a marked contrast to the facio-scapular form in which the shoulder muscles and upper arm muscles suffer chiefly. But in the case I refer to, the affection of the face is slight, and instead of it the hand muscles have suffered, in addition to those of the scapula and upper arm. She is a girl of twenty-three, the seventh of a family of eleven, all of the same sex, a fact to note. Three of them—the first, second, and fifth—suffer from a similar disease; three others died in early life; four are healthy.* Their parents were not

* Mr. H. Burland of Finedon, has kindly furnished me with some particulars of the family of the case last mentioned. Not only does it consist of eleven girls and one boy, but the mother was one of a family of twelve girls; all of the girls, except herself, died in early life. Of the patient's sisters, in one, now thirty years of age, difficulty in walking was first noted at fifteen after measles, weakness of the arms only at twenty-seven. She can now walk only with much difficulty. Movements of the hips and knees are weak, so are flexion and eversion of the ankle, but inversion is strong. The knee-jerks are absent. The flexors and extensors of the hands and fingers are weak but not much wasted. The upper arm muscles are small and feeble, but not the deltoid. The trapezii, serrati, and latissimi are all wasted. The

related, and no like disease can be heard of in the family. Yet the affection of four sisters shows how such a vital tendency may be congenital in a family, though not inherited.

In the patient difficulty in raising the toes was first noted at sixteen, and weakness in the arms at eighteen. The affection of the shoulder girdle and upper arm was precisely that of an advanced case of Landouzy form. The notes state that "little trace remains, in substance or in power, of the trapezii, serrati, latissimi, or pectorales. The upper arm muscles are thin and feeble, especially on the right side; the deltoids retain some substance. Equally great, however, is the affection of the distal muscles. We find no trace of the supinator longus. The flexors of the wrist and fingers retain some power; the extensors hardly any. Even the interossei are shrunken and very weak; if the first phalanges are passively extended, the others can only just be straightened by an effort. The metacarpal bone of the thumb can be well bent back, but no extension of its phalanges is possible." Everywhere, I may say, electric excitability was diminished with power, and alike to each current, as is the rule in myopathy. Thus in the upper limbs the condition was that of the scapulo-humeral form, but there was an unusual degree of affection of the distal part of the limb.

In the legs the affection was almost limited to the muscles in front of the lower leg. The movements of the hip and thigh were good and strong, and the knee-jerks active. Thus the

clavicular part of the sterno-mastoid is good. The orbiculares palpebrarum are feeble, the frontales strong. In the other sister, now twenty-five, weakness in walking was noticed at thirteen, after sore throat. In her the frontales have no power and the orbiculares are weak, and both parts of the sterno-mastoids are small. The muscles of the shoulder, elbow, and hand are all good, but there is some impairment of free movements. In the legs there is very little weakness, but the knee-jerks are absent and there is much inco-ordination. The nature of this case is thus doubtful.

affection of the legs was purely distal, as in the boy I have shown you; the proximal muscles were normal. In the arms the affection was both proximal and distal. Yet these two cases are linked together, and connected with other forms by the common features I have mentioned.

Pathology and Treatment.

I have spoken of all these cases as myopathic maladies, as muscular abiotrophy, the result of a defective vital endurance, inherent in the embryonal tissue from which the muscular structures of the body arise—a defect variable in distribution, in character, and in tendency, but essentially the same in all. In a few cases trifling changes have been found in the nervous system; in most it has been absolutely normal, both nerves and central structures, even by the latest methods of research, and in cases of most prolonged duration and profound degree. This fact seems effectually to dispose of the hypothesis that the muscular affection is in any way the result of the trifling changes that have been found in a few cases, or the more considerable atrophy of the spinal nerve cells found in one case. When we remember the fact, which I pointed out in the last lecture, that abiotrophy is met with in the nervous system, in various structures, and of various course, it is strange that it is not more often met with as an associated state. It is not surprising, moreover, that slight secondary changes should be met with in the spinal cord when there has been prolonged secondary spinal curvature, with all its effects on the circulation. Such trifling changes, met with in a few cases, irregular in seat and distribution, can have no real significance. Yet they have been thought to suggest that some day it may be discovered that the muscular condition depends, after all, on a morbid state of the spinal cord. This idea is a relic of the old fancy, for it was

nothing more, that there are in the spinal cord special trophic centres for the muscles apart from the motor cells of the anterior cornua. It is a doctrine that should have died of inanition long ago; purely hypothetical, every definite fact is opposed to it.

The vitality of the muscles presents a strange duplicity. Perhaps it is not so mysterious as it seems at first view; the same double relation may be seen, I think, elsewhere, although less clearly. The nutrition of the muscle depends on that of the nerves through which its function is called forth. If the nerves slowly degenerate, so does the muscle; if rapidly, from descending irritation, the muscles undergo speedy complete degeneration. Yet the muscle has a life which we may call organic, belonging to it as a structural entity, in consequence of which it may undergo morbid changes, apart from the nervous system, and may fail to live on, though the nerves preserve an unimpaired vitality. This failure is what I have called muscular abiotrophy—failure of nutrition from defective vitality—and for brevity we call it myopathy.

The treatment of this condition is a narrow subject, yet not unimportant. Its importance is positive and negative; to know what we cannot do and what we can. Life itself is beyond the influence of medicinal agents. Some degenerative changes seem to be within control, at least in slight degree, but it is otherwise with those vital tendencies which seem inherent. We can discern no clear evidence that they can be thus influenced; we can perceive no positive effect from the administration of medicine of any kind, and no faster failure when such agents are omitted. Yet even vital energy is not altogether beyond indirect influence. There is one agency, but only one, which has a sure effect on the vital nutrition of the muscles, and that is their voluntary use, their stimulation through the nerves with which their nutritional integrity is so mysteriously bound up. That is the lesson

taught by all true muscular growth, by the disciples of Sandow, as by every athlete. The application of the lesson to these diseases is all-important; it is seen alike whether it is enforced or disregarded.

Muscular exercise, adapted in degree to the muscular state, is the one agent which distinctly stems the ebbing tide of life, and hinders the failure of muscular strength and muscular nutrition. Neither electricity nor massage can exert more than a small fraction of the influence of voluntary use. Without it no definite effect can be observed from their employment; with it they seem sometimes to do a little good, and upward massage promotes the circulation in the muscles and the renewal of the blood plasma, from which their nutritional elements are derived. But that is all we can say.

When contractures or any accidental causes arrest the voluntary use of the muscles for even a few months, the weakness and the wasting increase rapidly, and speedily reach a degree from which there is no recovery. Contracture occurs readily in the muscles in which there is much increase of connective tissue, especially in the calf muscles, but the tendency to it varies. In some cases it prevents standing and walking long before this would be lost from the weakness of the muscles of the upper leg. Then the division of the Achilles tendons is imperative. I have mentioned * one case in which this contraction occurred so early, in such degree, that at five years of age the ability to stand would soon have been lost from this alone; tenotomy enabled the patient to walk until twelve, and then again became necessary. It preserved the ability for years; at twenty the patient could still walk three miles.

When the contraction occurs late, and the weakness of the muscles of the knee and hip is great, the question may arise

* "Pseudo-hypertrophic Paralysis," Case 35, and p. 55.

whether the rest the operation entails will not augment the weakness, so that the effect of tenotomy is useless. But the rest need be but brief, and its effect may be lessened by movements while lying. If the contraction is not extreme it may be diminished without tenotomy by an apparatus I devised some years ago,* in which elastic traction on leather around the foot is made from leather around the leg, the two being united by a narrow piece behind the heel; this bends, but has sufficient vertical resistance to prevent pressure on the skin, which, if continuous, so soon becomes intolerable. Worn during rest and indoors, the effect of the very gentle constant elastic traction is most remarkable.

At the knee and hip the contraction is of the flexors, and is the speedy consequence of posture which involves flexion of these joints as the patient sits or lies. It usually attends a degree of weakness which precludes any prospect of good from operation. It emphasizes only too strongly the importance of maintaining as long as possible the upright posture. When the patient can only sit, not only do these contractions of the hip and knee occur, but the weakness of the muscles of the trunk induces inevitably an inclination to one side, which leads to curvature of the spine, with all its consequences on the movements of the chest, and the disposition to chronic changes in the lungs by which most sufferers find the end of general life.

Elsewhere, in the arms especially, the essential element in treatment is the same persevering efforts to maintain the exercise of the muscles. For this gentle gymnastic exercises are extremely useful, especially the various apparatus for developing the muscles of the arms. But it is difficult to secure the necessary perseverance without the incentive which games provide. Fencing, indeed, is a most effective

* "Clin. Lect." ed. 1895, p. 194.

exercise when the failure of the arms is slight, if the exertion is moderate and the object kept in view. Cycling gives some exercise to the arms as well as to the legs, and a tricycle is often practicable and most useful when a bicycle cannot be ridden. Lawn tennis involves too severe effort to be commonly available, but its degraded indoor form of ping-pong necessitates the varied but gentle use of many muscles, which makes it far more than a mere game to sufferers from this disease, and it is additionally useful if played in the open air. When the movements of the hand begin to be impaired, the action of playing the piano is very useful, and the art should be acquired in anticipation of its need, that the pleasure should be an incentive to exercise, as it can scarcely be in the stage of laboured acquisition.

It is the case with all measures which merely hinder a progressive malady—their influence, however great, is unobtrusive. It is negative and not positive. How real and important it has been is only perceived when it ceases. The speed with which the malady then advances reveals the effect that had been unnoticed. The sufferer and his friends are inapt to realize these negative results, and prone to think that no good is done because no improvement is perceived. We may do something to save them from this error, and we must be careful not to fall into it ourselves. We should also do our best to try to save them from efforts to obtain good from the many straws held out for hope to grasp at, which are magnified by promise into rods of rescue. Those who promise with assurance that which cannot be, always find too ready credence, as is often discovered only too late.

LECTURE V.

METALLIC POISONING.

Delivered at the National Hospital for the Paralysed and Epileptic, on October 22, 1901.

Gentlemen:—Metallic poisoning has occupied a considerable amount of public attention during the last year. The epidemic of arsenical poisoning caused by the contamination of beer, and the consternation it produced, will be fresh in your minds. Many of you may also have noted the efforts which have been made to lessen the amount of lead-poisoning incidental to the process of glazing pottery, and the difficulty there is in practical prevention. Cases of lead-poisoning that occur in local trades appeal to the public by their concentration and by the apparent possibility of their prevention. But that which is met with in such industries is but a trifling proportion of the whole amount in the kingdom, most of which occurs among those who are engaged in the work of renewing for us the freshness and fairness of our habitations—the house-painters.

Lead.

Here, for your observation, are two patients suffering from the effects of lead on the nervous system, and I wish to avail myself of their presence to impress upon you some facts regarding metallic poisoning. Some of them they illustrate, others they do not. Both patients exemplify the fact I have just mentioned—both are house-painters. One presents the

characteristic effect of the disease—wrist-drop and paralysis of the extensors in the forearms—and the other does not. I think I may assume that you are familiar with the features of this paralysis, and I need not describe them in detail. Some points will come before us presently. But the case to which I would first and specially direct your attention is that of the patient who has no paralysis. He can move his hands freely and well, but he is suffering from various symptoms of feebleness of the nervous system. He is weak generally, although, as you see, he is fairly nourished. He can only walk with difficulty, but there is some cause for this, as we shall see. He complains of numbness at times in the right foot and left thigh. He has had much headache, and when he holds his hands out they present fine tremor, to which we must return. His difficulty in walking is partly due to an injury long ago. In 1888 he sprained his right ankle so severely as to be in the Bristol Royal Infirmary for four months. Some weeks after his discharge he injured the instep on the same side. The injury seems to have led to disease, because he returned to the infirmary and some bone was removed. Contraction in the calf must have followed, for the Achilles tendon had afterwards to be divided. Subsequently a toe was removed—why we do not know. He slowly recovered and was at last able to resume work, but after working for some time he again lost strength and his left leg became thin. His weakness culminated in an attack of colic. This was six years ago; only after a long period of rest did he gain enough strength to resume work, with, after a time, a similar result. After another attack of colic a year ago, he was so much more feeble that he had to give up and has not worked since. During the last year he has also suffered from much headache and from the trembling of the hands. The weakness of the legs has also become so much worse that he was only able to walk with

crutches until he came into the hospital seven weeks ago. Yet apart from the tremor, there is little that is objective to correspond to his disability. His knee-jerks are rather active, but he presents no foot-clonus. His left leg is thin and his foot shows traces of the operations he has undergone. His muscular strength is small and movements are rather jerky and not sustained. When I first saw him he had been carefully examined and notes had been taken. I was told that it seemed to be a case of "simple neurasthenia." I looked casually at the bed-card, and at once my eye was caught by the record of his occupation, "Painter." I looked from the bed-card to his gums, and there I saw written in equally distinct characters the record of the effect of his occupation—in a conspicuous lead-line. Further inquiry elicited the fact of the two attacks of lead-colic which I have mentioned, one six years ago and one a year since, but also that he had never suffered from any degree of wrist-drop.

Let me interrupt my special subject for a moment, to consider the diagnosis that had been made. The history of the word "neurasthenia" is noteworthy. It is a contribution to medical nomenclature which we owe to our transatlantic brethren, and it attained universal use with the utmost celerity. The concise and concrete character of the word gives it a satisfying definiteness. This depends to a large extent on its classical and somewhat graceful sound. Not only is it graceful to the ear, but it is grateful to the mind of the patient who suffers and longs to know from what, who longs to have a name for that which he, or more often she, feels must be a more definite malady than is suggested by the common-place designation of "nervous weakness." How far its practical utility, which, if low, is definite, has influenced its use I cannot say. But its use has extended far beyond the needs of the patient, and indeed did so from the first. It has firmly established itself in current clinical

terminology. But it often tends to be too satisfying. Men are apt to rest on it as they would not on its English equivalent. If they do not actually think that they have found the malady from which the patient is suffering, an influence is often exerted on them of which they are unconscious, which lessens the tendency to go further in the search for the whole morbid state. Words are our servants, but they often exert a very masterly influence upon us, none the less effective because we are not conscious of it. They have also their own vitality, feeble or vigorous, and we have little power to influence their career. This fact has come distinctly within my own experience. I have to confess to the authorship of two words. One, "myotatic," was always a puny infant, and I doubt whether it still maintains an independent existence. The other, "knee-jerk," instantly attained universal use, and, indeed, I think has seemed to most persons to have sprung spontaneously from the thing itself, without suggestion—perhaps the greatest compliment a word can pay its author. But the general use at once achieved by "neurasthenia" was in spite of a strong objection to it which was felt by many. The Royal College of Physicians of London could not include it in their "Nomenclature of Disease," and yet it is now one of the most common of medical words in every language. It would be instructive in more than one way to have a careful study of the forces which have influenced its career, but that I cannot attempt. We must, I think, admit that not only is it a satisfying word to those who suffer, but it has a certain convenience which has almost compelled many to employ it who at first objected. If I may be pardoned for a partial paradox, its convenience is not the less real because this rests on features that are illusory. Remember that the word is a name which should have little meaning, even to those who use it. You may employ it to collect the symptoms of the case under a

general designation, but do not let it cover them as a cloak.

But we must come back to our patient. When we had ascertained the facts I have mentioned, it was impossible to doubt that the man was the subject of lead-poisoning or that his symptoms of nervous weakness had the same origin. They had developed after he had passed from the hands of the surgeons and had gained strength enough to resume work. On each occasion, it was after he had been at work for some time that he began to fail, lost strength, and suffered from the symptoms you have heard, and each relapse was attended by the colic which demonstrated the action of the toxic influence to which his work exposed him, and emphasised the relation of the renewed weakness to this influence. That this influence is the effective cause of his symptoms, little as they would themselves suggest it, is shown by his history, and it is confirmed in an instructive way by careful observation of his state, especially by two features. The tremor he presents, which I will ask you to observe carefully, is a fine tremor which occurs only on movement, but it differs from the tremor so often seen in hysterical and nervous persons. That is a general irregular movement of the hand and fingers. Here you will observe that there is a quick lateral movement of the fingers, the result of contractions in the interossei. This is a peculiar and unusual feature, suggesting a special cause. Tremor is well known to be a result of lead-poisoning, and you know that it is a characteristic of the toxic influence of another metal—mercury. The second indication that he is still suffering from the influence of lead on the nervous system is very curious. It has been before observed, at least in Germany, but opportunities for detecting it are rare, and are still more rarely utilised. You are familiar with the wrist-drop, which is a

common effect of lead, the atrophic paralysis of the extensor muscles in the forearm. A good example of it is presented by the second patient before you. Such wrist-drop is due to neuritis and is accompanied by the characteristic "reaction of degeneration" to electricity—loss of faradic and increase of voltaic irritability, and to the latter a change in polar response. In health, contraction occurs to a weaker current with the kathode, the negative pole, than with the anode, the positive pole. In the reaction of degeneration this order is reversed, the contraction occurs more readily with the anode. In this patient we found this polar change in all the muscles that would be affected in wrist-drop and in no others, although it was the only change in reaction that could be detected. All the other muscles of his limbs, including those of the legs, presented the normal order of response. I am sorry—I was going to say—that you cannot see this. He has improved so much during the last few weeks that, on one side, the order of polar response has become normal, and on the other side, is on the way to be so, for the response is now equal to each pole. When he came in, the lead in his system must have exerted a definite influence on the nerve-endings in these muscles; he must have been on the verge of wrist-drop. This condition thus affords us additional evidence of the present action of lead on his nervous system.

My object in showing you this case is that it may impress on you the great variety of symptoms to which lead may give rise, and the importance of not overlooking the cause in consequence of the equivocal character of the effects. The variety of disturbance of the nervous system which may be produced is very great. Indeed, you might be prepared for this when you remember how wide and severe are the effects of acute lead-poisoning, how gravely the brain itself suffers in some cases, manifested by what is called "saturnine

encephalopathy," often attended by optic neuritis. In more chronic cases the symptoms seem to depend on the momentum with which the poison acts, and on the predisposition which the individual possesses. Let me enumerate some of the effects which I remember to have met with. Lead may cause not only the common peripheral neuritis but a slower chronic atrophy of the muscles, seen first in the interossei; it is precisely like spinal atrophy, and probably such, but differing in that it does not progress if its cause ceases to act, although it is far more enduring than the wrist-drop; it may be permanent, though it does not increase. Lead may cause some forms of sclerosis of the cord, usually slight in degree; it may cause optic nerve-atrophy; and many forms of functional disorder may result from it. It may cause tremor, as you see in this patient; chronic convulsions, like those of epilepsy; and hysteria, with its varied manifestations, in predisposed subjects. Neuralgia, sometimes of great severity, may be due to it, and headache is a frequent effect, as well as the symptoms of general nervous weakness. In all such cases, in which there is nothing in the symptoms to suggest the cause, this may escape you, unless you are put upon its track either by the occurrence of other associated cases of lead-poisoning, by the occupation of the patient, or by the presence of its great sign, the lead-line on the gums.

Let me dwell briefly on the line, because thereon hangs one of my lessons. It is typical in proportion as it approaches Euclid's definition of length without breadth, always provided it is black. It is said to be blue, but I have never myself been able to see any colour in it. It is not always to be seen, and often it is not to be seen readily. You know, or should know, the reason why it may be absent even in the most pronounced cases. It is the edge of a deposit of sulphide of lead beneath the inner surface of the gum, where this is separated from the teeth even in a very slight

degree. The sulphur comes from albuminous substances which decompose there. Sometimes you see a similar deposit in the mucous membrane of the lower lip, when there is tartar on the teeth with which it comes in contact. Tartar contains organic substance mixed with earthy salts from the saliva, and it yields enough sulphur to act on the lead. We have not this deposit in the patients here to-day. But I should like you afterwards carefully to compare the line which is present in each. In the first it is conspicuous, but it is not continuous; it is in separate pieces, where the gums are more detached. The second patient has always been particular about his teeth and careful about cleanliness—hence the detachment of the gums is slight; and although the line is present everywhere it is extremely narrow and only seen on looking closely. Indeed, it was altogether missed at first. When looking for a lead-line use your ophthalmoscope lens, which I presume you are never without. Careful as this patient has always been, he must have gradually accumulated lead in his system, but his final breakdown, with the wrist-drop which you see, he attributes to a cause which it is well to note. He ascribes it, apparently with reason, to the work of painting some ceilings. In this work the man who paints has to stand below that which he is painting, and it is impossible for him to avoid inhaling some of the spray which his brush necessarily throws off—spray which may be loaded with lead. It is the most perilous form of house-painting. I wish that the rich who indulge in the luxury of painted ceilings knew at what price it has to be paid for, not by them, but by those who produce the special beauty they desire. I think that house decorators ought to make the fact known to those who ask for this superfluity, for such it certainly is.

But I have not quite done with the lead-line. Far more common than the rare cases in which it is absent, are

those in which it exists only in fragments, often much smaller and fewer than in this patient. It may be at only two or three isolated spots, or on the tips of the projections of gum between the teeth. It can then only be found by a most careful and thorough search, on the upper jaw as well as the lower. Remember that the smallest fragment, if distinct, is as significant as the most extensive deposit. A year or two ago a patient came to me from the other side of the world and said that he was suffering from muscular wasting. I found considerable atrophy of the muscles of the forearms, but it was of the muscles that suffer in lead-poisoning, with characteristic wrist-drop as its result. No others were affected. They had lost faradic irritability and presented very little voltaic irritability, but this is common after a time in such cases, if voltaic electricity has not been applied. The excitability to voltaism quickly improves in this state after a short course of treatment with it. The onset had been subacute, just as it commonly is from lead. Of course, I at once examined his gums. At first sight they seemed normal, but on more thorough inspection, and especially on examining the gums of the upper jaw, several spots were found at which there was a slight separation from the teeth, and there I found two or three indubitable fragments of lead-line. I afterwards also found some black points at the ends of the projections between the teeth of the lower jaw. I could ascertain no source for the poisoning. The patient was a grocer, but had not for years been accustomed to handle the lead-paper by which tea is protected. Still, I could not doubt that he was suffering from simple lead-palsy, and I therefore felt justified in giving him a very hopeful opinion, a prospect of steady although slow improvement under treatment. The forecast was entirely justified. He went home in a year's time almost well. But I should not have spoken so plainly, perhaps, had I known what he

afterwards told me. After I had expressed my opinion he said that he had been sent to England as a sort of forlorn hope. He had been told that he was suffering from progressive muscular atrophy which nothing was likely to arrest. His own words were that he started under what he "felt to be a sentence of death." Perhaps he exaggerated. It is always well to remember the advice once given by a wise physician: "Never believe anything a patient says that another doctor said." But if he had told me this before, I should have been less emphatic in a statement which was evidently a contradiction of that which had been told him. I should have given him some hope and should have allowed this to be gradually augmented by his experience. It is always desirable to avoid expressing a difference of opinion more strongly than is absolutely necessary. And I heard of it again; I heard of it from his country, after his return. But I cannot doubt that the cause of the palsy might have been discovered by a sufficiently minute search, and it is to impress the importance of this upon you that I tell you of the case. It is an illustration of one of the wise sayings which the late Sir William Jenner was wont, I might almost say, to stick into the minds of his pupils, "More mistakes are made, many more, by not looking than by not knowing."

But there is one last resort for the detection of this cause of palsy. If the symptoms are such as to suggest lead, and you are sure that there is no trace of lead-line in any part of the gums, you may feel confident that it is not at work, provided the state of the gums is such as would certainly give rise to it. If the gums are everywhere perfect you cannot feel thus sure. I have seen saturnine wrist-drop without a trace of line, because the gums were in perfect apposition to the teeth; there was no room for doubt; the case was one of the many which once occurred in Sheffield in consequence of the solvent power of peaty water upon leaden pipes. Yet

we have a source of information. If the case is seen early, its nature may be decided by an analysis of the urine, especially after iodide of potassium has been given for a few days. It is a rather troublesome process, needing a large quantity of urine, but it may be decisive. I remember employing it in one case of wrist-drop perfectly like that of lead in distribution, course, and character. The result was negative, and its negation proved only too trustworthy; the extensor palsy was followed by wasting elsewhere and by general, rapid, progressive muscular atrophy.

Arsenic.

I mentioned arsenic as another metallic poisoning which has loomed large in the public and professional eye. The extensive epidemic of arsenic-poisoning in the north has had at least one good result—a vast increase in the general professional knowledge of the subject, so that the facts which were previously familiar to a few are now known to all. But it may still not be quite useless for me to impress on you the need for fixing its signs on your mind, ready for revival, because the need for the knowledge may come when you least expect it. Like lead, it causes neuritis, but arsenical neuritis usually affects the legs before the arms, although the latter suffer in severe cases. Moreover, its effects on the nerves vary much; the common palsy may be absent, and the sensory nerves may suffer much or most, and hence the symptoms may be equivocal and even misleading—sometimes, indeed, they may be identical with those of tabes.

Just as the line on the gums is the indication of lead, changes in the skin, especially certain forms of pigmentation, constitute the outward and visible sign of the influence of arsenic, visible at least if you look for them, and a sign if

you know what they are like. They have been abundantly described, not only by Dr. E. S. Reynolds, of Manchester, to whose perception of their significance we owe the discovery of the cause of the epidemic, but especially by Dr. H. G. Brooke and Dr. Leslie Roberts, who have published a very full account of them and their pathology in the "British Journal of Dermatology" for April, 1902. I only propose to mention some salient points regarding the pigmentation, points that have come frequently under my own notice. Other changes are also common, especially the thickening of the skin of the palms and soles, but the pigmentation is that which is likely, as a rule, to attract attention, and is of most practical importance. Moreover, this Hospital is the only place at which you are at all likely to become practically acquainted with it. Arsenic alone enables us to prevent or lessen the troublesome eruption which bromide causes, and few patients who take the combination for more than two years escape pigmentation. In one case I was able to discern the precise amount which had been effective. The darkening of the skin was first distinct when the patient had taken liquor arsenicalis for two years in uniform quantities, and the total was 104 grains of arsenious acid, or one grain weekly. Doubtless slight signs of it might have been discerned sooner. But I think that if you look at the neck of many old cases of epilepsy attending here, you will meet with an example before long. The change is most marked on the skin of the trunk, front and back, but it extends to the limbs and also to the neck, where indications of it may be detected with least inconvenience. Given with bromide, arsenic has very little tendency to act upon the nerves, even when a sufficient amount has been taken to cause extreme pigmentation. I have a strong impression that it is most readily produced in those who are most prone to the bromide acne, but the impression must of necessity remain such, because the

disposition to the acne leads to the administration of larger doses of arsenic.

The impunity with which arsenic can be taken with bromid, and the peril that attends a far smaller amount in beer, is very remarkable. The only explanation I can offer is that alcohol augments, and bromid restrains, the metabolism in the nerves and therefore the tendency for arsenic to replace the phosphorus and impair function and nutrition.

Whether or not you are able to become acquainted with the aspect of the skin that is due to arsenic, it may serve you in good stead if you remember its occurrence and also its chief features. The brown pigmentation begins as small spots, which may commence, as I have seen, in spots of congestive redness, and the brown tint succeeds the red. These dark spots sometimes exist alone, more often they are seen chiefly on the outer portions of the most pigmented regions, where the tint is more uniform. The uniformity may be due to coalescence of the spots, but sometimes this diffuse colouration seems the first thing. The tint is that which is called sepia, a warm sepia or cold sepia, according to its duration, to use the terms employed by artists. It is not so dark as the pigmentation of Addison's disease, and has not the same distribution. Although it is more marked at places at which there has been pressure, it occurs irrespectively of this and is greatest on the parts of the trunk least affected in Addison's disease. When the small discrete spots coalesce, they often leave small areas which are unpigmented and which have a pearly whiteness. Three weeks ago I saw a patient whose skin over the whole trunk was of a deep mahogany colour from this disease, with such white spots scattered over it. He was so prone to the bromide rash that even the dose of arsenic he was taking (seven minims with 25 grains of bromide twice a day) did not suffice to keep him

entirely free. But he had been free from fits for 16 months and his parents would not hear of a reduction in either the bromide or the arsenic. The darkening of the skin, they said, was quite unimportant, and that is the common opinion of those who present it when they know its cause. I have constantly to explain to patients its origin, and to give them the choice between the bromide spots and the darkening of the skin; there is never hesitation in choosing the latter. The white spots I have mentioned are common and somewhat characteristic, although they are met with in other forms of pigmentation. They have been called "rain-washed" spots from their scattered distribution, but their whiteness suggests a more potent agency than simple washing. I believe that their pearly whiteness is not the effect of contrast alone, but that they are actually paler than the normal skin. Why such areas should not only resist the pigmentation but should apparently lose what they possessed, is a mystery which the pathologists have not, I think, explained. But we may note that there seems to be a curious solidarity in these pigmentary processes, in consequence of which excess may entail adjacent deficiency and, I may add, a deficiency may be attended by adjacent excess.

Perhaps you will allow me to digress for a moment to mention to you a very remarkable illustration of the latter fact—increased pigmentation in the vicinity of its diminution. I have mentioned the case elsewhere,* but it will probably be new to you. It is that of a man who had traumatic meningeal hæmorrhage over the left hemisphere. As a result of this, during the three days he lived after the injury, the right, opposite half of the hair of his head and of his brown moustache and beard became blanched so as to be almost white. The change was watched during life and care-

* Manual of Diseases of the Nervous System.

fully noted after death. It was like that which has been described as the result of profound emotion, but it was due here to a physical agency. It can only be explained by assuming that the disordered innervation so changed the secretion at the roots of the hair as to produce a material capable of ascending the hairs and discharging their pigment. But after death we noticed another thing, which leads me to mention the case. The very grey, almost white, right half of the beard was separated from the brown left half by a narrow vertical line, or narrow zone, in the middle line, in which the hair had become almost black. Apparently where the disordered influence ceased in its extreme degree, at the blending of the innervation of the two sides, a change in the pigmentary process had occurred of the opposite character. Mysterious as the fact is, and perhaps impossible to explain, it illustrates the close relation between the *plus* and the *minus* in pigmentary processes. I am not sure that we may not find another illustration of this in some cases of atrophy of the pigment of the choroid adjacent to a spot at which it is collected, and also in the heaping up of pigment in association with areas of atrophy. But this may have another explanation.

To return to my proper subject. I desire especially to fix in your mind the fact of the occurrence of this characteristic pigmentation. Whether or not you are able to become actually acquainted with its appearance, it may chance to be as useful to you as it was to me last year. Last February a young Indian civil servant, a district magistrate, was brought to me by his family medical attendant. He had just arrived in England with symptoms of extensive peripheral neuritis, so severe that he could not stand or walk without assistance. There was much weakness of the legs as well as wasting of the muscles. There was incoördination and absence of the knee-jerks, considerable fibrillation in the

wasted muscles of the thighs, and great diminution in electrical excitability, which also presented a peculiarity with which I need not now trouble you. There was also considerable impairment of sensibility to pain and touch. His hands likewise were weak and very unsteady. The illness had followed a sharp attack of intermittent fever two or three months before, and he had previously suffered badly from the ordinary malarial fever in India. Such neuritis is well known as an occasional sequel to these malarial fevers, caused apparently by a toxin which is left in the system. But in that form the muscles of the lower legs are chiefly affected and incoördination is seldom a prominent symptom. When I came to examine the patient's body I found it covered with pigmentation perfectly like that of arsenic. Indeed, I could not doubt its nature. Of course, I questioned him carefully regarding any possible source of arsenic-poisoning, and especially whether he had taken any tonic containing arsenic. It seemed certain that he had not, and no accidental source could be ascertained. Thus the sequence of events suggested malarial influence as the cause, but it was impossible to misinterpret the declaration so clearly written on his skin of the influence of arsenic. Moreover, another fact was learned which gave support to the cutaneous message. About the time of the commencement of his illness he had an attack of shingles. It is a long time (35 years) since Mr. Hutchinson pointed out that herpes zoster may be caused by arsenic. Hence we were compelled to consider that his condition was due to this, in part or altogether; the facts were too definite, although inexplicable, and we treated the patient accordingly. Six weeks later the medical attendant wrote to me that the mystery was cleared up. News had been received from India of the discovery of a prolonged attempt to kill the patient and his family by the habitual introduction of arsenic into their food. Several members of the family

had suffered severely and the nurse had actually died from the poison. Our patient improved steadily, and I saw him a fortnight ago practically well, able to walk fairly and cycle 30 miles. The ataxy had vanished; some blunting of sensibility on the front half of the soles alone remained. He will soon return to his work in India. What is of particular interest, because it relates to a point about which we know little, is that the arsenical pigmentation has almost disappeared.

Another pertinent instance of the revelation which this sign may afford, I need only allude to, because I have already described it in my first series of Clinical Lectures under the heading "Mistaken Diagnosis." The patient came with what seemed to be characteristic symptoms of ordinary tabes—pains, ataxy, and loss of knee-jerk. He came when I could not properly examine him, so I ordered him some medicine containing arsenic and arranged to see him again in a month. Then when I stripped him to test his sensation, to my consternation I found his skin covered with typical arsenical pigmentation. He was a colour-merchant, accustomed to deal in various pigments, but he had also taken for a long time a tonic pill containing arsenic. Both these cases illustrate the tendency of the sensory nerves to suffer under arsenic far more than under lead. The patient recovered, and he taught me another lesson. There is evidence that iodide of potassium promotes the elimination of arsenic as well as of lead. The path by which it can pass away is, of necessity, the blood. You know that it is not well to give iodide freely to a patient with recent and severe lead poisoning, because the lead stored in the tissues may be thrown into the blood too suddenly. This case showed that we should be equally cautious in the case of arsenic. Iodide in very moderate doses led to such an increase in the symptoms of

irritation of the nerves as to compel its discontinuance for a time and very gradual resumption.

I have only one other instance to mention to you and that is an illustration of the other side of the shield. An epileptic patient, a young woman, about 30 years of age, presented marked pigmentation from the arsenic taken with bromide. From the latter she was deriving great good. I explained the nature of the darkening of the skin and she was satisfied. About six months later I received a letter from her sister written in dire distress. The aspect of the skin had caused two medical men to assure her that the patient was certainly the subject of Addison's disease—a progressive incurable malady. I could only reply that the patient when I last saw her presented darkening of the skin due to the arsenic. I heard no more of the case. It is right to say that this was before the Manchester epidemic had its opportunity of exerting its educational influence on the profession.

The facts I have given you are commonplace. Many of them may have been known to some of you, some of them may have been known to many of you; but one fact I am sure is true of all my hearers—for it is as true of myself as of anyone—the commonplace is, of all knowledge, that which we can least afford to despise or to disregard.

LECTURE VI.

SYPHILITIC DISEASES OF THE NERVOUS SYSTEM.*

Delivered at the Medical Graduates' College and Polyclinic, October, 1902.

Gentlemen:—I have been asked to lecture to you at short notice, but fortunately a subject has been assigned to me—syphilitic disease of the nervous system. I have not, however, been told what to say. The subject far transcends the limit of a single lecture. All I can do is to present for your consideration some general principles concerning the most important practical branches of the subject, those which should guide us in the prognosis and treatment of these diseases.

I fear I can only give you that which is old. But the old is not, as such, to be despised. It has been well said by Ribot, as to senile failure of memory, that "the new perishes, the old endures." The saying is true in a much wider application, although it is only a partial truth. We may also remember another aspect of the relation. To present old knowledge to new minds involves a constant renewal of its youth. The seed that is old produces a new plant. It may be true that "there is nothing new under the sun," but there is a sense in which it is also true that there is nothing which is not new. The rising sun is old enough, but each sunrise is a new event.

* "British Medical Journal," April 4, 1903.

Even in my restricted topic I must confine myself to the principles which seem to me of most importance. First, however, we must have a clear perception of what we are dealing with when we speak of "syphilitic disease" of the "nervous system." What do we understand by "nervous system"? If we only mean the actual nerve cells and fibres, the functional elements, I might paraphrase that chapter in a book on Iceland which Dr. Johnson said he could repeat from memory, the chapter on Icelandic snakes. Doubtless you know your Boswell, and remember that this feat of memory turned out to be the repetition of the sentence—"Throughout the whole of Iceland there are no snakes." So I might say, "there is no syphilitic disease of the nerve structures." But the term "nervous system" includes also the neuroglia, which supports and separates the nerve elements, the blood vessels, which penetrate and permeate the centres, and the membranes, which enclose and protect them. These structures are among the most frequent seats of the morbid processes of constitutional syphilis. The nerve elements proper do indeed undergo degeneration after syphilis, but this is a different condition. I will refer to it presently, but it is not within our special subject.

I have not made this comparison with the snakes of Iceland merely to enunciate a paradox. Behind it lies an important fact. Syphilitic disease of the nerve centres, developing in what may be termed the "adventitial" elements, produces symptoms for the most part through the changes it causes in the nerve elements themselves. But these are simple, not specific. They are secondary to the syphilitic disease, but they are the same as would be caused by any other disease of the same character, whatever its nature. The significance of this fact is far reaching. Specific treatment acts only on the specific process. It has no direct influence on the secondary changes by which the symptoms are produced. This

fact should be kept in view in all your practical conclusions, and to impress it upon you is one of my chief objects to-day.

Let me give you some illustrations. You may not need them, but, believe me, the art of getting the knowledge you have not, is to welcome that which you think you have.

To take first a salient example of the simple nature of the process by which syphilitic disease causes symptoms—consider syphilitic disease of a cerebral artery. The disease narrows the vessel, or narrows the orifice of a branch which arises at the affected spot, but causes hardly any symptom until a clot suddenly forms. The narrowing makes the flow sluggish in the "backwater" just beyond the diseased spot; at last, clot forms on the altered wall and quickly closes the vessel. It extends on, up to a branch from which a collateral supply can maintain the circulation; if there is none it spreads to the distal twigs. The region of the brain from which the blood supply is thus suddenly cut off, at once loses its function. Unless a collateral flow can be established in an hour or two, it perishes, undergoing necrosis, "necrotic softening," of necessity enduring, with persistent loss of function. But the thrombus which forms is simple, not specific, and so is the resulting softening. The only syphilitic process is that in the wall of the artery.

Reduce the disease of the artery to a cicatricial state as quickly as you can by treatment, you cannot change its effects. You can exert no more influence on the thrombosis or on the softening than in a case in which the softening is due to embolism. Nor can you influence the characteristic symptoms which are due to these simple effects of the disease. This must be clear to you, but it is not so many years since I heard a man of professional distinction say of such a case of sudden syphilitic hemiplegia, "Oh, he has a node within the skull; give him mercury and he will get all right."

Sometimes the course of the case seems to verify such a

favourable prognosis. Under due treatment the symptoms lessen and may pass away. They do so in the same manner and degree in many cases of embolism from heart disease, and do so whether medicine is given or not. The initial effects of any vascular lesion are much wider than those that are permanent, because the area of brain disturbed is greater than the area destroyed, and the permanent symptoms depend on the functional importance of the latter.

Take, again, the case of a syphilitic gumma growing from the membranes of the spinal cord. It slowly compresses the cord and this causes paraplegia. The effects on the cord are precisely the same as would be produced by any other kind of tumour. If pressure is slowly exerted, the fibres lose all power of conduction long before they suffer total destruction. Secondary degeneration may follow with all its symptoms, and yet certain elements of the fibres may retain such continuity as permits their ready renewal when the pressure is removed. Conduction, if it has not been lost too long, may be restored when treatment causes the compressing growth to shrink, just as it is slowly restored when a sarcoma outside the cord is removed by a surgical operation. But the treatment does good, not by its influence on the changes in the spinal cord, on which the symptoms directly depend, but by acting on the specific cause of those changes. These pass away if there has not been actual destruction.

Such syphilomata may form on the sheaths of the cranial nerves and produce the same effects—arrest of function, with palsy in the part supplied, recovering when the compressing agent is removed. It is so, also, with the similar processes in the membranes of the brain. But here, as in the nerves and the membranes of spinal cord, the processes of growth are associated with another process—that of inflammation.

The subject of syphilitic inflammation brings before us some pathological conceptions which it is important to dis-

cern. The characteristic specific process is neoplastic, the formation of tissue which, when recent, is semigelatinous in aspect ("gummatous"), and resembles, histologically, granulation tissue. After a time it becomes firmer, and two changes occur. Its elements become transformed to fibres, which contract in a cicatricial change, or they undergo fatty degeneration and caseation. This caseation occurs in several discrete spots, perhaps because the vessels of the neoplasm become thickened and closed. These areas may coalesce, but do not constitute the general uniform caseation so characteristic of a tuberculous mass. This caseation in several foci, combined with the gummatous aspect of the recent parts, is the most characteristic feature of these specific growths.

Every process of tissue formation, whatever its nature, involves vascular disturbance proportioned to its acuteness. Syphilomatous tissue always forms rapidly, and the attendant vascular disturbance constitutes a state of active congestion, and even of inflammation.

Thus we can trace the two elements, the inflammatory and the neoplastic, in all syphilitic processes. In proportion as they are acute the inflammatory element preponderates. We meet with foci in the meninges, which we should call inflammation, but for the amount of gummy tissue produced, which may be such that in places the characteristic spots of caseation are seen. We may be then in doubt whether to call it a gumma with adjacent inflammation, or inflammation of a gummatous character, and it is really a choice of terms. But it is doubtful how far the inflammation itself is specific or is simple.

Inflammation is a curious process. You know how it spreads in some structures when once lighted up. We sometimes see this in the peritoneum in a terrible degree. In the cerebral membranes an inflammation once excited, by whatever cause, tends to spread swiftly in proportion to

its acuteness. In proportion to this, the inflammatory element predominates in syphilitic meningitis, and in that proportion it has the features of simple inflammation. It speedily becomes extensive, general, and even may be bilateral. It seems as though some form of morbid agent had the power of exciting inflammation which at once attains a degree far in excess of its cause. It seems also to be less amenable to treatment in proportion to its acuteness, in proportion to the degree in which the simple inflammation runs away, as it were, from the specific cause. We meet with instances of this, happily not often, in the cerebral membranes. We meet with the same, more frequently, in the spinal cord.

Acute myelitis, focal or transverse, is met with so often in the early stage of constitutional syphilis that it is impossible to doubt a causal relation. Yet the inflammation presents the same features, and runs a course quite the same, as in patients in whom syphilis can be excluded. Remember that in all acute inflammations of organs, the proper functional structures undergo rapid destructive changes which are regarded as a participation in the inflammation. But these are simple in aspect, similar in every form of inflammation equally acute. They are intense in proportion to the inflammation, and may speedily attain a degree from which recovery is slow, imperfect, or impossible. This is conspicuous in these cases of acute myelitis, and they justify the inference that in these cases also the symptoms depend on the simple effects of the specific element. It is alike true whether the inflammation is purely such, or whether the damage to the nerve elements is brought about by disease of the arterial walls at the spot affected. In the more chronic forms of local inflammation, in which the process passes into that which may be termed sclerosis, the same fact is apparent. The morbid process is interstitial, and the nerve elements suffer secondarily.

Local chronic inflammation of the cerebral cortex has other effects which are also due to the simple damage to the nerve elements in the cortical substance. Even if the effects of inflammation pass away, no structure is ever quite the same as before, and in the grey matter of the brain a slight residual alteration may entail a grave change in function. Instead of stimulated, proportioned liberation of nerve energy there is spontaneous discharge which gives rise to local convulsion. This may occur from the irritation of the active inflammation, but also as a residual effect, such as might be due to the inflammation caused by a blow. It also is a manifestation of simple changes on which specific treatment can have and does have no influence.

The points I have mentioned are not matters of theoretical pathology; they are of the utmost practical importance. On your perception of the mode in which the symptoms are produced depends the correctness of the forecast you can form, of the prognosis you can give. The essential point is that the specific process can be influenced by treatment, but this can produce no effect on the simple changes on which the symptoms directly depend. The forecast is favourable in proportion as the latter can pass away when their cause ceases to act. Hence it is important that you should cultivate the habit of picturing to yourselves what is the exact morbid state, and strive to discern the nature of the changes which give rise to the symptoms. Endeavour to acquire the habit of forming such a mental picture. Obtain all the data you can, and then strive to see with your mind's eye, and trust your vision. It may seem difficult at first, but will become easier with practice. It will often serve you in good stead in many forms of disease, in many parts.

Prognosis.

The application of these facts to prognosis should be self-evident. The prospect of improvement and recovery depends on the extent to which, after the removal of the specific disease, the simple processes on which the symptoms depend can pass away. In all tissues the tendency to recovery is great, if the change has not proceeded to actual destruction. Thus you will perceive that in arterial obstruction there is a speedy, permanent destruction of tissue, and function can only be regained in so far as it is capable of compensation by the other hemisphere, or in the degree in which initial damage exceeds actual destruction. In the case of slow compression the possibility of recovery depends partly on its degree, and still more on its duration. Pressure always involves secondary inflammatory changes in the compressed tissues. These go on to cicatricial processes if the pressure has been long continued; in time they constitute a grave hindrance, sometimes an insuperable obstacle, to the restoration of continuous structure and conducting function. Paraplegia from such pressure may be complete for a month, and recovery will probably, in time, be perfect. If complete for three months, recovery may possibly be perfect, but is more likely to fall short of the normal state; but if complete paraplegia from compression has endured for six months only partial recovery can be expected after even the complete removal of the cause, and after a year the return of function may be very slight, and at best will not be great. Of course, these are but rough assertions; they are, I believe, approximately true, but they are given chiefly to fix on your minds the importance of considering duration in the attempt to forecast the future of a case. You have doubtless long since learned by experience, that may have been painful and certainly must have often been trying, the urgency with which

a patient, and still more the friends, desire to know the future course of the malady. They expect from us a prescience more than human. It seems reasonable to them that we should be able to see into the future with the same certainty as into the past, and the same confidence as into the present. We are fortunate when the conditions are such that we can give them that which they ask for; but you will only discredit yourselves in the long run by holding out confident hope when it is unjustified. Strive, then, to perceive the morbid state, and if you cannot anticipate much improvement, it is seldom that you cannot hold out hope of some, but take care that you are clearly understood, and that your "may" is not construed as "will."

It may seem strange to some of you that any cases of this character should lose their chance of complete recovery by reason of their long duration before appropriate treatment is employed. But it seems to be a law of Nature that everything that never should be, sometimes is. One cause is because some patients suffer long before they seek advice. It is not so with paralysing maladies, but it is so sometimes with those that damage nerves, and even with arterial disease so placed as to cause only slight symptoms for a long time before arrest of the circulation is produced, and when the parts affected are those to which no motor functions are related. It is not often, now, that such disease is undetected in consequence of the ignorance of the doctor who is consulted, but it happens sometimes because he does not make a sufficiently careful search to discover slight but conclusive symptoms of organic disease. Among these optic neuritis is one of the most important and most frequently overlooked. Indeed, I may say in passing that no defect in medical education seems so persistent as ophthalmoscopic training. It will be so until the inspection of the normal disc is part of practical physiology, or at least is taught with the stethoscope at

the beginning of practical medicine. Clinical work abounds with unused opportunities, because the eye is never looked into until the end of the student's course.

Another frequent reason for the prolonged unchecked development of the disease is a belief that it cannot be. One of the great lessons of life—sometimes only learned late—is hesitation in pronouncing anything impossible. Certainly, this disease is sometimes met with under conditions which suggest that it is impossible, especially to those who know the patient best. One fact, at any rate, you should always remember. It can be excluded only when there has never been exposure to possible infection in the common way. If there has been, if it has been escaped by "luck"—its denial should have very little influence on your opinion. You know also how constantly its occurrence in married women has no symptoms to make it suspected, and it is important to remember that the evidence afforded by children may be absent. Indeed, the subject is one in which it is important never to forget the rule—a rule of almost universal application—that the absence of evidence is of very little value compared with that of its presence. Nature is unveracious as well as man, or rather seems so because our ears are tuned only to its affirmations. Its "no" is often altogether untrustworthy, but this is because we interpret its silence as negative without the least justification for doing so. Almost every common symptom of a morbid state is sometimes absent; every indication of a common cause is sometimes absent. Hence assurance is possible only when the common cause can be absolutely excluded.

Thus the practical lesson which follows from the perception of these facts is that the cause of the symptoms of the various syphilitic diseases which damage the nervous system is a secondary result of the specific process. Remove the latter by treatment, remove it as completely as possible; the secon-

dary effects may pass away, or may only lessen, or may persist with only slight diminution. A cicatrix from a gumma in the liver may be unimportant; in the nerve centres it may cause enduring symptoms. In the cortex of the brain organic epilepsy may be its result; in the deeper centre, permanent palsy. Even a gumma pressing on the cord may be removed by treatment too late to prevent a degree of mischief which cannot pass away, and some palsy may be enduring.

Remember also that the actual result of treatment on the specific process depends on its duration. The new formation rapidly diminishes in size under treatment. Apparently the tissue elements that are of recent formation, chiefly cellular, undergo granular disintegration, the results of which are quickly removed; but the older cells undergo a change into fibres, which may perhaps also be formed between them, and the diminution in size is partly due to the progressive cicatricial contraction of this fibrous tissue, which proceeds to such an extreme degree. Treatment seems to promote this cicatricial process in the tissue elements which have attained too mature a stage to undergo disintegration and total removal. But this process has its own consequences; that which it entirely surrounds suffers inevitable constriction, which may perpetuate or even augment the effects of the active disease. This is another reason why your prognosis in cases that have proceeded unchecked for some time, should be very cautious.

In a previous lecture, I pointed out that this change, going on all round the wall of an artery, seemed to have induced its occlusion, and that this effect had apparently been promoted by the influence of the treatment which arrested the disease and helped the cicatricial change. Happily such annular disease is rare, whether in a nerve or in an artery, and the conducting function of a nerve may be slowly restored in spite of considerable enduring constriction.

Yet the impairment may continue long after cicatrization of function is established. This fact seems to be incomprehensible to many. As long as symptoms continue, as long as the normal state is not regained, it is believed that there must be specific disease unconquered; treatment must be made more energetic, must be repeated. Iodide is continued and increased; course after course of mercurial inunction is employed. Sometimes all is to no purpose; sometimes there is the slow natural recovery we know so well when causal disease has ceased, a recovery then inevitably assigned to the persistent treatment employed, although it may be, I believe, in spite of it. You may ask, Is not this reasonable? Are we not justified in our inference, since we cannot see what is going on?

Well, I would ask you to consider what you do in other conditions that are beyond the direct range of observation. You infer from that which you can directly discern the character of the process you cannot actually perceive. If a patient comes to you with a recent node on the tibia you know that under iodide or mercury it will have disappeared in six weeks. If the node has existed for six months, at the end of six weeks it will have become much less, but periosteal thickening will still be felt, and this will be very slow in disappearing. It will lessen in time, just the same whether or not you continue specific treatment. The residual thickening is due to cicatricial tissue, which must persist after all the specific element is removed. So with a syphilitic ulcer of the skin. At the end of six or eight weeks' treatment—if this is adequate—all the peculiar specific thickening is gone; the ulcer may not be healed, but all that remains is a simple ulcer undergoing simple cicatrization, which will go on just as well if you do not continue specific treatment as if you do.

When the ulcer is healed, what remains? A scar, first livid, afterwards brownish, in which the skin is thinner and

smoother than elsewhere—a scar which remains to the end of life. In every tissue it is the same. Every morbid process leaves its changes in proportion to its degree and character. A destructive process leaves cicatricial tissue in place of normal structure, and this may even surround a cavity if there has been a massive destruction. Simple inflammation may leave a slighter change, but usually such as to be discerned, at least by the microscope, and this degree is colossal compared to the changes in finer nutrition which must always ensue. The effect on function, even of the slightest residual change, is enduring, and important in proportion to the delicacy and differentiation of function of the structures affected. Hence, whenever the nerve structures are damaged by specific processes, we should be prepared for imperfect recovery, however successful our treatment of the primary process may be. It is indeed remarkable how great is the restoration of function in many cases, in the course of time. But the fact I wish to impress upon you is that the "time" is not occupied by the removal of the specific disease, but by the recovery of the damaged structures when this has been removed.

You will perceive from what I have said that the pathology, prognosis and treatment of these maladies are closely connected, or rather they are the same, viewed from different sides. You can only frame a forecast by discerning whether the causal disease can be removed completely, and how far the nerve elements can recover. For this purpose it is essential to form a mental conception of the process I have mentioned. When you cannot see with the eye you must strive instead to see with the mind. The great difference between various processes will then rise clearly into view, and all that the difference entails.

For the most part the symptoms of these processes are hardly ever really *slow* in their production. They are sub-

chronic, subacute, acute, or sudden, reaching a considerable degree in less than three months, and often in much less. Of all it is true that the more rapid the development of the symptoms, the less is their course affected by specific treatment. Those which are most deliberate are chiefly the result of a gumma, compressing or irritating, or of gummatous inflammation of the membranes. Over these, treatment exerts its greatest influence, and in them the prognosis is the best, subject to the limitations I have mentioned—long duration and irritative influence. The acute symptoms, acute as distinguished from sudden, are for the most part the result of inflammation. This, although excited by a specific cause, varies in the relative amount of inflammation proper, which is greater the more acute the process. The nerve structures suffer chiefly from this, and the more rapidly the symptoms develop the greater is the destructive implication of the nerve elements, the less is the room for the influence of specific treatment. We see this strikingly in the cases of acute transverse myelitis, so frequent in syphilitic subjects, in whom, as I have said, it runs a course differing in no respect from that which it pursues in other patients. The cases of actually sudden onset present a course over which specific treatment can have and does have no influence. I need not repeat what I have said regarding this point.

Treatment.

It may seem strange that the therapeutic progress of the last half century has left us without any new remedy for syphilis. But we need not be ashamed of the nineteenth century, since it has produced the second of our two certain remedies, iodide. Its value was discovered in the thirties, and in England. Like the majority of our most useful drugs, its service was found by empirical observation, indeed, it may

be said, almost by accident. The real or supposed influence on goitre of burnt sponge, led to the discovery that it contained iodine, and to the use of combinations of iodine for other swellings, among them for nodes. From this the step was small to the discernment of its power over other syphilitic processes, a power as certain as it is mysterious.

The older remedy, mercury, has come down to us from the dim distance of a thousand years. Its use by the Arabians is said to have been anticipated in India, but the Orient shrouds in impenetrable mist the beginnings of all things. Its service in syphilis is believed to have been one of the Eastern lessons the Crusaders brought back to Europe, unless it had been conveyed by the Moors to Spain with skill in other arts, such as the Alhambra retains for us.

The two agents stand on a different level. We have other facts regarding the power of mercury which make its influence on syphilis less strange. It is possible that it has a capacity of arresting many diseases that depend on an organismal virus, although it is only in a slow disease that we can avail ourselves of this effect. The fact was ascertained in the early days of the Brown Institution that an animal under the influence of mercury was unaffected by a dose of anthrax virus which would certainly be fatal to a similar animal not so protected. It is believed to have an influence on all processes of inflammation. Those of our profession who have occasion to watch the only internal inflammations which can be directly observed, those within the eye, are firm in their belief in the influence of mercury upon them, irrespective of their specific nature. I may add that it has been employed in most of the cases of cerebral meningitis, including some apparently tuberculous, in which recovery has been known. This is, at least, true of my own experience. Mercury is a poison, but a considerable quantity may be contained in the blood, if gradually introduced, and we can understand that

this may be sufficient to be fatal to the organisms of disease long before it is fatal to the living elements of the body. But to permit this effect the disease must be one of slow development.

We can thus at least seem to understand the influence of mercury in syphilitic processes, and also the trust, placed on it in the early stage, when the organisms must be in their first flush of growth in a fresh soil. But iodide has no such toxic property. It is a salt not far removed in nature from common salt, yet its influence on the processes of syphilis is certain. This must be due to the iodine, and it is said to be possessed in equal degree by the combination of iodine with a vegetable oil, "iodipin," or "iodinol." Iodide, in itself, seems almost inert on the normal processes of the body, and yet it has a profound influence on this morbid process. But we must remember that all these elementary bodies seem able to enter into combination with albuminous substances, modifying their influence, sometimes profoundly. A slight change in constitution will transform a food into a poison. The mystery of the certain influence of iodide is made more salient by our uncertainty as to its effect in other morbid states. It has been, and is, thought to do good in many; but, if we search for stringent proof, we shall find ourselves in agreement with Nothnagel, who, in his great book on "Materia Medica," says: "The therapeutic influence of iodide is undoubted only in one condition, tertiary syphilis, and it is possible in hyperplastic affections of the lymphatic glands and thyroid. In all other conditions its influence is doubtful and uncertain. We have employed it much, very much, but in other maladies we have never obtained definite and incontestable certainty that the improvement and recovery occasionally observed were due to it."*

* Nothnagel and Rossbach, "Arzneimettellehre." 7te Aufl., 1894, p. 299.

Of course, its administration in many maladies must often coincide with spontaneous improvement, so very easily ascribed to coincident treatment. But when due allowance is made for this, and when the cases in which it has failed are given their weight, does the lesson of the residue differ much from the experience of Nothnagel? The contrast presented by its power over syphilitic new formations is most strange. Not less so is its innocuous character compared with the toxic power of mercury. It is difficult not to believe that the future has some revelation in store for us, which may not only explain what is now mysterious, but in doing so may augment our resources, alike in means and method.

It is not through diseases of the nervous system that any new means of combating syphilis are likely to be discovered. Duty precludes us from the attempt. The effects of such disease are so grave, and are rendered graver by every day's delay, that we dare not pause to search for a new agent. We must employ at once the means we know and trust, and leave the task of discovery to those whose province is less profound. As a fact, moreover, nothing is such a hindrance to the discovery of new remedies, truly such, as the efficiency of those we have. If we know what will cure, we seldom dare search for another remedy.

The difference between the two agents is familiar and important. Iodide can be thrown into the system in any quantity; mercury can only be introduced gradually. For the chief lesions of constitutional syphilis they seem equally useful. But when the process is inflammation, we should expect mercury to have the more certain influence, and observation affords some confirmation of this. It is not for a physician to express an opinion on the treatment of the early stage of the disease, but in this there is a consensus of opinion of the need for mercury on the part of those who can speak with authority. Regarding the later processes, there seems

to be a general feeling, rather than opinion, that mercury can do a little more, and can do it more thoroughly than iodide, but that the latter does its work more speedily. The feeling deserves respect, even practical respect, but I do not think it rests on strong evidence—at least so far as the nervous system is concerned. The opinion is largely due to the use of mercury after iodide has lessened the symptoms, and probably after the syphilitic process has been entirely removed. The damaged nerve structures have slowly regained function during the subsequent mercurial treatment, to which the later improvement has been assigned. No delicate therapeutical inferences ought ever to be drawn from these diseases. We are right to apply to them all we can know or suspect, but the pitfalls of fallacy are, as we have already seen, many and treacherous. Real knowledge can only be increased from that which is within the range of vision, from the surface of the body. It rests with the dermatologists to augment our certainty, and increase our power. They can see and we cannot. The risks that delay involves are to them trifling. They should endeavour to eliminate every source of fallacy, to distinguish between the specific and the simple elements in the processes they treat, and to make their therapeutic observations precise, multiple, and widely known. Then we should feel sure of much we now only suspect, and perhaps we should doubt some things we now believe.

Of one thing I am quite sure, that the symptoms of a syphilitic growth may diminish and pass away as rapidly if iodide only is given, as we can conceive possible. I do not affirm that it is always so, but I remember many cases in which 10 gr. three times a day produced, in a week, cessation of headache and distinct diminution of early optic neuritis, which had vanished at the end of a fortnight. In one such case slight hemiplegia had disappeared at the end of a month, and hemianopia, which had been complete, was reduced to part of

one lower quadrant. But this remained permanent in spite of mercury also.

It is said that much larger doses of iodide are sometimes necessary, but I have not myself met with evidence of this, except in patients who have long been taking moderate doses. The use of large doses—30 gr. or 40 gr.—has been called the new American system. I have already referred to the saying that "there is nothing new under the sun." Many years ago I had occasion to go through some of the old casebooks of University College Hospital, and I found one case in which Dr. John Taylor gave 40 gr. three times a day for constitutional syphilis, somewhere about 1847. The same physician, by the way, at the same time, made constant use of the thermometer at that time to ascertain the presence and degree of fever, long before the instrument came into general use.

I think it is not wise to give iodide and mercury together in full doses, except for a short time in a very urgent case, when every means and measure are needed to stem life-threatening disease. There is reason to think that iodide promotes the elimination of mercury, as it does of most metallic substances, and thus hinders the retention in the system of enough to act upon the processes of the disease. We cannot be sure that an adequate quantity is present in the blood unless we have such evidence as is afforded by slight inflammation of the gums. The difference in the readiness with which this is produced is probably due to differences in individual power of elimination, which seem to be effected chiefly by the liver.

This explanation is, at any rate, plausible, and I think useful. It suggests that, if there is not the sign in the gums of enough mercury in the blood to act on the tissue processes, we should increase the dose—double it, treble it—until we obtain the evidence. But it is wiser not to give at the same time an agent which has the power of promoting its elimina-

tion, except there be the urgency I have referred to. All experience shows that the affection of the gums is evidence that enough is present in the system. It may then be stopped for a day or two, and continued in smaller quantity to maintain the effect for three or four weeks; then the iodide may wisely be resumed. It will have its own effect, and may also at first maintain the influence of the mercury by bringing into the blood that which has entered the tissues.

We cannot improve on the physicians of long ago in the method of administering mercury. The object is to get enough in the system, and this can only be slowly achieved, or inconvenient toxic symptoms result. One of these is gastro-intestinal irritation, which is always greatest when the administration is by the mouth. The old method was to rub it into the skin, and it is, I am sure, the wisest. One modern method is to inject it under the skin. Whether or not this is equally effectual, which I doubt, it is certainly much more uncomfortable than inunction. By the latter the gums can be affected in five or six days, which is as speedily as the result can safely be obtained by any system.

Inunction is much facilitated by the use of the oleate, which is not only cleaner in aspect, but, I think, surer in result. Order a drachm of the 10 per cent. oleate to be rubbed in twice a day for three or four days, and then continue once a day until the end of a week. If the gums then show no sign, resume the two daily rubbings until they do. But remember one small precaution, which I should be ashamed to mention to you did I not know how much depends upon the trifles of our work. See that the same small piece of flannel is always used, and let the first inunction be 2 drachms. Otherwise the nurse will think it so much cleaner and nicer to take a fresh piece of flannel each time, which will retain more than half the dose, and you will wonder why your patient's gums show no sign.

I do not think it matters into what part the mercury is rubbed. It is often convenient to have it rubbed in as near as you can to the affected part—close to the scalp if it is the brain, down the back if it is the spinal cord. This has a certain reason, that the oleate makes the skin rather sore, and thus effects slight counter-irritation. It has another reason, of a kind not to be despised. There is nowadays too much knowledge, public and popular, regarding the use of drugs and their purpose. Many of the laity, not alone of the male sex, draw an immediate inference from the use of mercury, an inference which is undesired by the patient. It is prevented when the site of inunction is near the seat of the disease, and when the colourless oleate is employed, instead of the blue ointment with its suggestive tint. Such a device for the avoidance of distress does not merit any deprecation on the ground of high morality. If it is true that "things are not what they seem," it is equally true that they sometimes should not seem to be what they are, when the seeming can do no good and may do harm.

One element in treatment I have yet to mention. I have left it to the last because it runs counter to the cherished practice of many of those who have had experience in the treatment of this disease. It concerns the duration of treatment. Regarding the processes of the constitutional disease, definitely such, my conviction is that specific treatment should be energetic, brief, renewed, but not continuous. By "brief" I mean that it should stop at the end of eight weeks or so, and be renewed after two, four, or six months, and that the patient should have three or four weeks' treatment with iodide every four months during the first year after any true specific symptoms, and every six months for the next three or four years.

Let me tell you the reason for my conviction. I have said it before, but a teacher who hesitates to repeat, shrinks from

his most important duty, and a learner who dislikes to hear the same thing twice over, lacks his most essential acquisition. Many years ago a patient came under my care with symptoms of a cerebral syphiloma. Under 10 gr. of potassium iodide three times a day the symptoms rapidly passed away, and the patient seemed well. He continued, however, to attend the hospital and to take the medicine. At the end of three or four months he presented symptoms of disease of the upper dorsal region of the spinal cord. They progressed rapidly, and caused his death. It seemed impossible that the disease could be syphilitic, since he was still taking the medicine which had so speedily removed all indications of such disease in the brain. But the necropsy shook our confidence in the seemingly impossible. In the brain there was the shrunken relic of the gumma; in the spinal cord, invading it from the membranes on one side, was a similar growth, in the active stage, typical under the microscope, and typical also in the naked-eye feature of disseminated caseation, a character of conclusive significance.

The lesson this case taught me I have since seen confirmed by other cases, not my own, however. It is that treatment may lose its power if continuously maintained. It is not altogether unintelligible that this should be. We cannot doubt that syphilis is an organismal disease, that it depends on bacilli as yet unidentified. We have yet no definite objective acquaintance with these microbes, but the symptoms of the disease harmonize best with the theory that their life is continued by germs which persist in various parts of the body when the organisms die. The course of the malady suggests that these germs may long "lie low" and latent, but develop from time to time under influences we cannot discern. A consideration of the course of the disease and its relation to treatment, compels the conclusion that this has no influence whatever on the latent germs. It seems to destroy the

developed and developing organisms, and thus dissipates the morbid processes they excite. But the most thorough treatment may fail to prevent future development of the disease, at whatever stage it is adopted, however early. I do not say that it has no such preventive effect; but instances have been met with by every one who has had much experience, in which there was recurrence after recurrence, in spite of most thorough treatment. The only reasonable explanation of this seems to be that the germs are indestructible. They cause no symptoms until some influence induces and permits their development. After a given outbreak there may be none left, and treatment then seems to have cured the disease. In another case there may be many, and then recurrence occurs after the same treatment. Hence, whether syphilis is or is not incurable as a constitutional malady, it is certainly one of the cure of which we can never be sure.

Hence my belief that the essential principle of treatment is that it should be energetic but not continuous. Energetic mercurial treatment is, of course, such as to affect the gums, and this cannot well be continued for more than six or eight weeks, nor need it be. Iodide can be given persistently in large doses for many months with no grave constitutional inconvenience, and I know that such long continuance is thought by some to be an essential element of thorough treatment. But the germs seem able to become acclimatized to the presence of the iodide and to develop in spite of it. The fact is not unintelligible. Dallinger found that a temperature which surely killed certain organisms, failed to hinder the vigorous development of their germs, if successive broods were gradually inured to heat, which was increased by slow degrees. So we can conceive that the syphilitic germs may gradually become accustomed to the presence of iodide. Even then, it is true, a great increase in the dose may arrest their development. But the increase cannot proceed indef-

initely, and the question is, Is this long-continued treatment needed? My own conviction is that after eight weeks of adequate treatment the specific disease is at an end. All that remains is the non-specific element in the process, or, especially in the case of the nervous system, the secondary simple effects in the nerve elements, which can only pass away slowly, and sometimes cannot pass away completely.

Thus the nerve elements suffer simple changes in consequence of the specific process adjacent to them, changes which are not directly influenced by the treatment of the latter. In the same way they may suffer simple degenerative changes, not as the direct effect of adjacent specific disease, but as a mysterious sequel to constitutional syphilis. These also are not influenced by specific treatment. They are the post-syphilitic or para-syphilitic degenerative diseases, of which locomotor ataxy and general paralysis of the insane are the most frequent forms. They are primary degenerations, such as we know to be caused by chemical poisons, organic and inorganic, and they are probably due to a toxin, the result of the previous presence of the organisms of syphilis. Similar effects may follow a more acute organismal disease—diphtheria. We cannot doubt that the relation of these maladies to syphilis is similar, that the constitutional disease induces a perversion of some part of the chemistry of the body so that a poison results, perhaps with the help of other morbid influences—a poison that endures long afterwards. The evidence is purely that of sequence. In the case of diphtheria, which may cause symptoms identical with those of post-syphilitic tabes, the mechanism has been definitely proved, and this analogy gives great weight to the evidence of sequence. But, just as it is not pathological, so it is not therapeutical. Most tabetic patients have iodide given, and hope is held out of benefit, but the cases

are very few in which even slight improvement can be traced. A large majority are sure they derived no benefit, and not, a few are certain that it did harm. The same is true of mercury, except that energetic treatment seems more often to be productive of distinct deterioration. We should not, indeed, expect evidence from treatment. The only sure antidote to diphtheria has no influence on its paralytic sequel.

But you should know that these toxinic degenerations sometimes develop before the true constitutional disease is at an end, while gummata and gummatous inflammation still occur, for which specific treatment is needed. The important fact is the combination of symptoms produced and the difficulty of discerning their causes. In early distinct tabes, paralysis of ocular muscles may occur, which may be of the nature so common in tabes, or may be due to a syphiloma of the nerve or specific neuritis. Still more perplexing are the cases to which Erb's attention has lately been specially directed, in which specific spinal pachymeningitis has coincided with true tabetic degeneration. If the nature of syphilis is such as I have suggested—if germs are deposited in the system which develop from time to time under influences of which we know nothing—it is easy to understand that the organismal processes, truly syphilitic, due to later germs, should coincide with degenerations which are the effect of toxins, the result of earlier broods.

Let me remind you, however, of one useful criterion of the existence of post-syphilitic degeneration which is often of service when you are puzzled by symptoms which may be due to either process. This is the isolated light-inaction of the iris—"isolated" because it still acts readily on accommodation. You are familiar with this, which is known by the name, as just as it is cumbersome, of the "Argyll-Robertson symptom." You know its association with tabes and general

paralysis as degenerative sequelæ of syphilis, but you may not know that it is met with in association with other conditions, and also as an isolated symptom. Even then, it is of great significance as an indication of old syphilis, and I have many times had reason to appreciate the guidance it has afforded.

But these degenerative conditions are not within my present subject and I have reached the limit of time at my disposal. I have endeavoured to put before you some general principles which should be kept in mind. If you strive to apply them to cases that seem clear, you will find it easier to use them in cases that are obscure. It is necessary to accustom yourselves to analysing the symptoms in simple cases, and discerning the grounds on which the prognosis is founded and the treatment based, in order to obtain the ability to do so in cases that are complex. Even those that seem most difficult will generally yield to a deliberate effort to apply to them the principles I have given you; they will yield, if not altogether, at least in a degree sufficient for immediate need. Moreover you will, I believe, seldom fail to find that your conclusions are confirmed, and your treatment justified, by that to which we have all to look,

"The balance in the hand of time."

LECTURE VII.

INEVITABLE FAILURE.

A STUDY OF SYPHILITIC ARTERIAL DISEASE.

Delivered at the National Hospital for the Paralysed and Epileptic, October 22, 1901.

Gentlemen:—Patients sometimes die when we expect them to live. The discrepancy between our forecast and the fact is, of course, the result of our ignorance. It is occasionally impressed upon us in a special manner when death terminates the reticence of life, and we are permitted to read the record of disease, and yet cannot solve the mystery. Sometimes, however, a careful scrutiny reveals to us the reason for our error, and when it is so, we can often learn many lessons. It is to an instance of this that I desire to direct your attention to-day. One of the lessons that it teaches is alike startling and unexpected. It is the evidence that the best and wisest efforts to prevent death probably promoted its occurrence, and could not but do so. They did not cause it, for it was, by the nature of things, inevitable, although the malady was such as is usually counted curable, and commonly is so. These are the facts of the case.

A girl, aged 25, a waitress, was admitted, after having suffered for three months from headache, which was severe from the first, and was referred to the front and back of the head. After it had existed for a month, vomiting was added to it, and both symptoms continued. Towards the end of

another month she was noticed to become strange in manner, and the mental disturbance increased, so that she is said to have been "always talking nonsense." Soon afterwards, three weeks before admission, some loss of power of the right side was noticed, which was thought to have commenced suddenly, but it subsequently increased. She also had a fit of some kind. The only other fact of importance is that a doctor, who knew something about her but does not seem to have treated her for this illness, stated that she had probably had syphilis. She was an ill-nourished, anæmic girl, very weak, and in a condition of stupor. She did not speak, or do what she was told, although she could evidently see those about her, and resented examination. There was considerable right hemiplegia, weakness of the right side of the face, complete paralysis of the upper arm, although she could move the forearm and hand a little; power in the leg was limited to slight movement of the foot. The left limbs were moved freely. There was also foot-clonus on the right side; sensation was impaired in the right limbs but not abolished; the optic discs were normal.

Those are the essential features she presented. Inunctions of mercurial ointment were ordered, a drachm twice a day. For a few days there was distinct improvement: she moved the hand better, and became able to answer questions. It was not, however, greater than we often see from the first influence of the rest and suitable food a patient receives in the ward of a hospital. The improvement soon ceased, and she became duller and feebler. At the end of a week 15 grs. of iodide of potassium, three times a day, was added to the mercury. But in a few days more the increase in weakness was so distinct that the inunctions were stopped, the iodide being continued alone. She continued in much the same condition for another fortnight, the stupor indeed increasing, and the movement of the right hand becoming less.

She had several slight convulsive attacks, some affecting only the right side. The optic discs continued free from any trace of neuritis. At the end of three weeks after admission, paralysis of the left side came on almost suddenly, complete. The stupor deepened to coma, the temperature rose to 104°, and three days later, on the twenty-sixth day after admission, she died.

The symptoms suggested a rapid cerebral growth. The prolonged headache, afterwards associated with vomiting, was especially significant of this. The course of the symptoms before admission was that which is met with from an untreated syphilitic tumour. Sudden hemiplegia is occasionally met with in the course of a tumour, especially towards the end, so that this fact—the alleged suddenness of the onset—did not seem to have much weight against the indications afforded by the other symptoms. Nor was the absence of optic neuritis a negative symptom of much significance. Optic neuritis is an incident in the course of a growth; it may occur at any period, or be absent. It is important to remember the general diagnostic law that the absence of a common symptom has small negative importance compared with the value of the positive indication which its presence affords. At the same time normal discs are unusual in a tumour of such rapid development, and therefore had rather more significance than they would have had with symptoms of more chronic course.

Here was a case in which it was reasonable to anticipate that treatment would have been successful, if the disease were of the nature assumed. Grave as was her state when she came in, it was far from hopeless, and yet the patient died. What solution of the mystery did death afford? The disease was found to be such as it was thought to be, so far as its nature was concerned, but it was not of the character supposed. It was syphilitic, but there was no growth. There

was extensive disease of the arteries, such, alike in extent and in degree, as is seldom seen. The disease was most intense at the commencement of each middle cerebral artery, at the place at which the passage of the anterior cerebral from the internal carotid converts the latter into the middle cerebral. Here on each side the disease had entirely surrounded the vessel. The external prominence was very slight, but the thickening of the wall had encroached on the cavity of the artery, even to the point of obliteration. It was especially great on the left side, and on this the anterior cerebral was likewise involved in the disease for some distance and also closed. In each middle cerebral the most intense disease extended, lessening in degree, for about one-half to three-quarters of an inch, but beyond this there were scattered spots of disease, nowhere surrounding the vessel. Similar patches were seen on each posterior cerebral artery. The posterior communicating arteries were almost free. The left anterior cerebral, after it had turned back above the corpus callosum in the inner side of the frontal lobe, was smaller than the other and pale, but perhaps only in consequence of contraction from the lessened blood supply to be presently mentioned. A similar change was found in the small branches which this and the middle cerebral send into the substance of the brain. To this condition I must return.

What effect had this arterial disease produced on the circulation within the vessels? The right middle cerebral was occupied by a recent clot, which extended from the disease at its origin throughout the whole vessel. It had completely closed it in its entire length, and there was commencing necrotic softening in the region of the brain supplied. This thrombus must have been the cause of the final hemiplegia—the palsy on the second side, the left, which came on three days before death, and caused it. The aspect of the clot corresponded to the duration of the paralysis; it

was red, uniform, and not adherent. The state of the brain supplied by the vessel was that which we should expect three days after the arrest of the blood supply. The disease at the origin of the artery appeared to have quite closed it, but some of the contraction may have been due to the influence of the agent—formalin—in which it had been at first placed, since some blood must have passed through until three days before death, when the clot formed.

In the other middle cerebral, the left, a remarkable condition was found. There was the intense disease at its origin which I have mentioned, thickening the wall, and encroaching on the cavity from every side, so as completely to close it. Similar disease extended along the wall for three-quarters of an inch, gradually diminishing but continued as scattered spots. Where greatest, it had so completely closed the vessel that there was no room for clot, but adjacent to it was an old coagulum occupying the vessel for about a third of an inch. It was decolorised, adherent, its aspect such as would correspond to the period since the first attack of hemiplegia, which you will remember was on the right side, opposite to this vessel. But the obstruction of the artery was complete, and the hemiplegia was incomplete! Beyond this thrombus the artery contained fluid blood, and there was no softening of the region of the brain supplied by it, such as usually follows the obstruction of an artery, and such as we found commencing on the other side, from the recent thrombosis in the opposite artery. The circulation had evidently been maintained in the distal part of the vessel. How had this occurred, so as to prevent the extension of the clot? The answer is strange. On this side an artery arose from the posterior cerebral and passed forward, entering the fissure of Sylvius, to join the middle cerebral just before its bifurcation where the fissure divides It was a vessel of some size, although far smaller than that which

it joined. It was free from disease, and had carried enough blood into the middle cerebral to prevent the extension of the thrombus, although of course far less than would have been received by the usual channel. But it was enough to prevent necrotic softening and complete hemiplegia, although it was not enough to prevent some paralysis and some instructive pathological changes, as we shall presently see.

Still more strange is it that another abnormal vessel had in like manner maintained the circulation in the anterior cerebral. I told you that this artery was involved in the disease which existed where it arises from the internal carotid, the disease at the beginning of the middle cerebral. The anterior cerebral was also completely closed, and adjacent to the closure was an old clot, decolorised and adherent, like that in the other vessel, and extending also along it for about one-third of an inch, just beyond the anterior communicating artery. Further on the vessel contained fluid blood, as did the middle cerebral beyond the clot in it. The reason was clear. Your anatomical lore should not have been so much attenuated by time as to make it needful for me to remind you that this anterior communicating artery completes the arterial circle at the base of the brain which you know by the name of our great countryman, Willis. This communicating artery is usually single. Here, however, it was double. A second artery crossed about a third of an inch in front of the ordinary vessel, and was equally large. The clot in the anterior cerebral extended beyond the first communicating artery, but not quite so far as the second, and through this the blood supply had been maintained in the distal part of the anterior cerebral, just as was the case in the middle cerebral. As in the latter, the amount of blood brought to it thus was much less than the normal flow, but it was enough to maintain the movement of the blood, to prevent the extension of the clot, and to prevent necrotic softening.

These facts enable us to understand the symptoms better, and also their mechanism. The limited clot in the left middle cerebral, adjacent to the occluding disease,* must have caused the onset of the right hemiplegia, by the sudden diminution in the blood supply to the motor region, although the abnormal artery from the posterior cerebral carried enough blood to prevent the paralysis being complete. In the same way the clot in the anterior cerebral may be assumed to have caused the mental symptoms, although its effect was lessened by the supply of blood brought by the second communicating artery. You know how often disease or injury of the anterior lobes, and especially of the left, has caused symptoms of this character.

But this does not enable us to understand the progressive increase in the symptoms, the increasing palsy, the deepening speechlessness and stupor. Farther careful examination discloses to us the apparent cause of this—indeed, I may say the certain cause. Extensive change is to be seen in certain parts of the cerebral substance. It has been carefully ascertained by our pathologist, Dr. Collier, but you will be able to observe it for yourselves. In most of the region of the cortex supplied by the middle cerebral on the left side, and much of that supplied by the anterior cerebral—the vessels we have just been speaking of—there is a very peculiar alteration. The grey substance is shrunken; it is diminished to not much more than half its normal thickness. Both this and the white substance beneath present a peculiar granular aspect and definite induration. Wherever this exists there is found considerable change in the small arterial twigs, which pass into the brain substance from the branches of the main

* The clot probably formed before the perfect closure by disease, when the aperture had become so small that very little blood could pass through. The complete occlusion immediately ensued.

arteries. The alteration in these arterioles is very definite. They are contracted, small and pale, evidently in consequence of the diminished blood supply, since you know that arteries adapt themselves to their contents. But Dr. Collier finds in them also a process of endarteritis; there are active changes between the internal and middle coats, cell proliferation thickening the middle coat, and still further diminishing the cavity. It is doubtful whether this endarteritis is syphilitic in nature; indeed it probably is not. A similar process has been found to be the mechanism by which the arterioles are obliterated wherever there is a considerable persistent diminution in the supply of blood. But we can be quite sure that this process has been progressive; it has gone on until death, in spite of treatment, apparently as a simple process—the result of the diminished blood supply.

The alteration in the structure of the brain must be ascribed to this change in the arterioles, slowly increasing the effect of the diminished blood supply, since the two coincide in distribution. Both are processes which have gone on of necessity, once set up by a persistent cause.

It should now be clear why the symptoms bore so much resemblance to those of cerebral tumour. The severe persistent headache, afterwards with vomiting, was the result of the morbid process in the vessels, extensive and unchecked. The disease must have commenced some time—certainly several weeks—before the headache began. Pain in the head is an inconstant symptom of such disease, but it often precedes, for a few days or weeks, the occurrence of thrombosis, which, with the sudden palsy it produces, is commonly the first proof of the disease. The headache is very rarely attended by vomiting, so conspicuous here. Such suffering almost invariably brings the patient under treatment; it is seldom that the disease progresses unchecked, but the girl had dosed herself with various things—phenacetin, antipyrin,

etc.—in the hope of relieving the pain, and apparently had no friends to secure proper treatment when she became incapable of doing so herself.

The second character by which the symptoms resembled those that would be caused by a growth was their steady progress, the increase of the right-sided weakness, of the inability to speak, and the mental dulness, in spite of her treatment in the hospital. We must ascribe this to the slow changes in the cerebral nutrition due to the causes we have just considered. Both it and the narrowing of the arterioles were secondary effects of the occlusion of the large arteries at their origin. The collateral supply of blood, which saved the patient from the severity of the sudden symptoms, was insufficient for the normal circulation in the arterioles or for normal nutrition of the brain substance. It was insufficient to prevent the slow development of other symptoms, at last equally severe, and not unlike those that would have been caused by a tumour, or might have been suddenly produced by extensive initial thrombosis. From that she was saved by the abnormal arteries. Such abnormal vessels are rare, but not extremely rare. They are, however, most unlikely to be present in any individual case. We can never assume their existence, still less their situation and influence. We cannot allow their possible existence to determine our diagnosis. This we can only base on the probable, and rely on the principles that guide us aright in the majority of cases. Yet our rules are always open to correction and improvement, or at least they should be.

As I have said, the case is a rare one; but no cases can teach us more which is important for common use than those that are rare. They touch the common on every side; they almost compel thorough study, and this involves the perception of their general and wider relations. Let us see what lessons we can gain from this case; if we secure some, we

shall learn, unconsciously perhaps, much more. In the attempt to point them out, and especially to be clear, I shall have to repeat myself. I ought not indeed to apologise for this, for as brevity is said to be essential to wit, so repetition is certainly essential to effective instruction.

I will begin with the question of the diagnosis of the disease. You know the saying, "It is easy to be wise after the event"; I may add that it is not easy to be wiser. If we met with a quite similar case we could be accurate in our diagnosis—easily. But Nature is not given to repetition. To be wiser we must learn lessons that we can apply under conditions not the same. We must not rest content with the perception that error was here inevitable, but could henceforward be avoided in identical circumstances. From every case we should endeavour to learn that which may tend to keep us right, whatever be the features of another case of the same nature. The lessons we may gain from the facts of this case are especially instructive, because they afford an instance of the way in which one symptom influences the significance of another, and of the need for comparing them, of weighing them, not only alone but together, in order to discern the resultant of their several forces, whether the mutual effect is increase or diminution.

The first important fact is the unusual duration and severity of the general cerebral symptoms, which must for two months have been the result of the arterial disease alone. It is important to note this, because had the fact been part of our common knowledge—the fact that such disease might have caused these symptoms—it might have caused more weight to be given to the alleged sudden onset of the right hemiplegia. Instead of this being regarded as compatible with a growth, it might have been considered to be strongly suggestive of vascular disease. The second fact is that progressive symptoms of local organic disease, uninfluenced by

treatment, persisting and increasing in spite of it, may be a secondary effect of arterial disease. This also is not part of our common knowledge. The conditions which led to it here are, of course, most unusual, but the same result may be met with under conditions less exceptional. The recognition of this fact might have caused more weight to have been placed on the absence of the optic neuritis, which, as I have said, usually attends a growth of such rapid course. It is difficult to say whether these considerations would or should have made the existence of a growth unlikely. We must not be too "wise after the event," even if we strive to be wiser.

There still remains the chief problem, Why did the patient die when it was thought that she would live? To be right or wrong matters little in itself; to prevent death matters everything. Without successful treatment, and except as subordinate to it, accurate diagnosis is as "thistledown without seed." In this case, it is true, the diagnostic error could have had little influence; it concerned only the special form of the disease; its nature was correctly recognised, and the misconception made no difference to the treatment that was right and was employed. But why did proper treatment fail? The answer brings before us considerations of great importance. We know but little of the way in which treatment acts upon syphilitic formations, acts on them so surely, induces their rapid diminution in size, and their ultimate change into cicatricial fibrous tissue. We must infer from the quick reduction in size of such growths that the formation of the softer cellular elements is arrested; that they undergo a process of disintegration, and the products of this quickly pass away. Those elements that are changing to fibres, or are actually such, are apparently incapable of such destruction. They seem to persist, but to shrink and contract, a change which goes on, as we see in every cicatricial

process, so that the ultimate result of a gumma is a firm nodule of fibrous tissue, such as may sometimes be met with in the liver. Note that the contraction of the fibroid elements is, and must be, an essential part of the process.

Such disease of the larger cerebral arteries is generally on the side of the wall only; it does not surround the vessel. Its disappearance under treatment is therefore only attended with a fibroid change on one side of the wall, unimportant unless a branch arises there which may be closed, or unless the substitution of extensible tissue for the elastic and contractile elements of the wall permits aneurysmal dilatation to take place. The ultimate condition caused by such disease, even with effective treatment, is one great cause of intracranial aneurysm. But, on the other hand, if the disease entirely surrounds the artery, the effect of the contraction of its tissue must be to lessen the calibre of the vessel. This is an inevitable result, serious in proportion to the degree of disease, to the degree to which the wall is thickened. Fortunately such a condition is rare—it is rare for such disease to surround a vessel—but you have a striking example of it in these arteries. The growth entirely encircles each middle cerebral artery and each anterior cerebral; the thickening of the wall is such as apparently to obliterate the cavity of the vessel. On each side—on the left against the old thrombus, on the right where the recent clot commenced—the vessel seems to be closed. In neither can we discern any persistent channel. There must have been one until the final occlusion occurred, but the opening must have been very small. We have here evidence which seems irresistible that the process which treatment was intended to induce—the process by which the morbid process lessens and ultimately cicatrises—could not hinder, and perhaps promoted, the contraction of the tissue in the wall of the vessel, so as to diminish the reduced calibre arteries, and on

the right side to induce the formation of the clot which was the cause of death. This is a startling fact, but from it I see no escape. Another indication of the process of contraction is afforded by the slight degree of external prominence of the disease in the larger arteries. Such disease, untreated, always projects considerably from the wall of the vessel. The absence of this prominence we must ascribe to the contraction induced by the month's treatment. Contraction of growth in the wall of a tube lessens the external projection, but at the expense of the cavity. Thus we are confronted by the fact that, with disease of this character, so extreme and so placed, the treatment by which it was hoped that life would be saved, and health restored, could not avert, if it did not accelerate, the gravest consequences of the disease.

It is difficult to arrive at any other conclusion than that death in this case was inevitable. The opportunity for effective treatment had passed before the patient came here. We cannot doubt that if she had sought medical help in the early stage of the malady, and had received that which was wise and adequate, her life would have been saved. We must, however, remember that in patients of her sex it is often difficult to discern or even suspect the specific nature of such disease. Sometimes it is scarecly possible for any suspicion of this cause to arise. Not long ago I saw a patient in whom a morbid process of this nature seemed quite out of the question, and to no one was it more inconceivable than to a doctor who had known her from childhood. She had certain spinal symptoms, chiefly in the arms, which seemed only to be accounted for by an increase in syringomyelia or "central gliomatosis," such as sometimes occurs soon after adult life is reached. But a very unexpected revelation disclosed to me the possibility of the constitutional influence we are discussing, and made it possible, and even probable,

that the symptoms were due to cervical pachymeningitis. The saying that "knowledge is power" received a perfect illustration, for the treatment which the knowledge entailed resulted in the complete removal of the symptoms, and they were of most threatening gravity.

The case was an illustration of another fact of which we are sometimes apt to lose sight: the absolute difference which exists between the improbable and the impossible. Of course there are degrees of probability and improbability. The extremely improbable is very near the impossible; the two may seem almost alike and appear to be on the same level when we look along the surface of things; but between them a deep gulf is fixed, a gulf unbridged. The impossible cannot be; the improbable, however unlikely, may be. In the course of life you may meet with cases in which it will be useful to remember this.

But I would have you, when you look at these arteries, to ask yourselves, What can be anticipated in such a condition? You will perceive from them, more forcibly than from words, that no therapeutic measures can alter the clot which has formed in the diseased vessels. A clot in a vessel is one of the most stable, most enduring, of morbid states. When thus closed, the cavity of an artery cannot be restored, nor can the effects of its closure on the brain substance be prevented or materially lessened. The symptoms of such closure, indeed, do commonly lessen, as do those which are produced by the embolic closure of an artery; they diminish because they are at first more extensive than correspond to the actual destruction of brain tissue. If iodide is given the improvement may be, and often is, ascribed to its influence, although without adequate ground.* By suitable means we may support and steady the circulation, and probably

* See the preceding lecture.

increase such collateral supply of blood as may be possible, but the utmost we can effect is very little.

Yet you should give iodide, unquestionably. The absolute necessity for treatment should be clear to you when you look at these vessels. Consider the wide extent of the isolated areas of disease, on the posterior cerebrals as well as the other vessels; hardly one is free. The disease which exists is always more extensive than corresponds to its obtrusive effects. Symptoms seldom bring us face to face with disease of the kind until an artery, or at least a branch of some importance, has been closed by thrombosis. The effects of this we cannot change by treatment, but we can never tell how much more disease there may be, disease we can remove, nor can we tell how near such disease may be to the closure of a branch or trunk. Such possible effects we may prevent by prompt measures, and I have seen cases in which such treatment seemed clearly to prevent the gravest results. Yet treatment has to be carefully adjusted to the patient's state. In this case the mercury had to be discontinued on account of weakness, and the fear of further enfeebling her heart. It was wise, and even wiser than was then discerned, because the risk of a fresh thrombosis was more imminent than could be perceived. On account of this danger it is not well to give, in such cases, the very large doses of iodide of potassium that are sometimes employed. Iodide is credited, as you know, and I think with reason, with the power of promoting coagulation of the blood, and has been employed for this purpose in aneurysm. Hence very large doses may facilitate the formation of a clot in a narrowed vessel. The dose which the patient had—15 grs. three times a day—is large enough, as I have many times seen, to influence such disease as rapidly as is reasonably possible, if the patient has not already been taking it, and is as much as it is wise to give under the circumstances.

Let me end by looking back at our starting place. This was the fact that we sometimes fail to save life when we expect to succeed, even when we have reason for our expectation. Such failure is among the sources of the distress which the incidents of our work so often cause, especially to young practitioners. But the facts of the case we have considered should fix in your mind the truth that even the full knowledge, which so seldom is within reach in ordinary practice, may demonstrate that the failure was inevitable, that the end could not have been averted by any means we could have used, or delayed by any effort we could have made. Thus it was here, and the same is often true when it cannot be ascertained. Therefore, do not let the discrepancy between your early forecast and the later fact, cause you to give way to misgivings which may be groundless, and regrets that must be vain.

LECTURE VIII.

SYRINGAL HÆMORRHAGE INTO THE SPINAL CORD.*

Delivered at the National Hospital for the Paralysed and Epileptic, February 8, 1903.

The human frame is the most complex and elaborate structure known to us. It is not surprising, therefore, that its development should sometimes fail to achieve completion, or that this failure should occur in the most elaborate of all its parts, the central nervous system. Defective development is found most frequently in the spinal cord, and it is to one effect of this imperfection that I ask your attention to-day. The precise defect we are concerned with is that which entails the presence of peculiar cavities in the cord. Such cavities have been ascribed in some cases to other causes. To this I will refer presently, but it is certain that they are often, and perhaps generally, the result of a local arrest of the process of development.

You know that the spinal cord is, at one stage, an elongated mass of embryonal tissue, having a medial furrow, the sides of which ultimately close. You know that the closure may fail to occur in part, and that even the closure of the bony canal may not take place, with "spina bifida" as the result. You know—I hope that I am not presuming on too much knowledge—that the defect in the bony canal may be slight or even absent, and yet it may be definite in the

* Lancet, October 10, 1903.

spinal cord. You may also know that there may be defective development within the parts that do coalesce. Embryonal tissue persists where its development into nerve structure fails to occur, and often there is an actual cavity in this tissue. Thus we may have cavities where closure has failed to occur, with or without adjacent embryonal tissue, or we may have cavities where persisting embryonal tissue has broken down. Those are the essential facts of what is called "syringo-myelia." There may be a cavity in the middle line, usually continuous with the central canal, where should be the posterior medial raphe. This is obviously due to imperfect closure. Or there may be a cavity adjacent to the posterior horn, often one on each side, but unequal in extent. These may be continuous with the central canal on one side or on both.

These lateral fissures in the posterior columns are especially important from a pathological point of view. In the process of normal development, a portion of embryonal tissue becomes isolated, and remains, unchanged, as the gelatinous "caput cornu posterioris." The lateral cavities apparently result from an abnormality or imperfection in this process. All the developmental cavities occur in the posterior and central regions of the cord,—a fact that is intelligible, since this region is the last to attain structural completeness. But such cavities, when dilated, may compress and damage the anterior horns, with muscular atrophy as the result, and even the lateral columns of the cord, causing spastic paralysis of the legs.

It has been maintained of late that similar cavities in the spinal cord sometimes result from preceding hæmorrhage, and Schultze* has suggested that their origin may

* Zeitschrift f. Nervenk., 1902. See also A. Westphal, Arch. f. Psych., 1903.

be minute hæmorrhages in the grey matter caused by difficult birth. This is a possible cause, although not yet proved. But their precise origin does not concern us now. It is their presence and not their cause with which we have to do.

The developmental nature of these cavities is often suggested by the fact I have mentioned—that they are bordered by undeveloped embryonal tissue. It forms a bounding layer, sometimes encroached on by erosion when liquid has long distended the cavity, sometimes considerably increased by a process of growth. Such residual tissue is prone to increase and its growth is occasionally so considerable as to constitute what is called "gliomatosis," *i. e.*, a glioma which can be traced to exuberant morbid development of such residual tissue. The word gliosis has been applied to it when it does not form a distinct tumour. The normal neuroglia is the remains of this embryonal tissue, and gliomata elsewhere have been thought to arise from spots of such residual tissue. It is certain that glioma of the pons—that infiltrating growth which causes great enlargement before it interferes with function—is sometimes connected by tracts of similar tissue with gliosis around a congenital cavity in the cervical spinal cord. This is a pregnant fact. It supports the hypothesis that such tumours may have their origin in residual undeveloped embryonal tissue. It shows us also that these developmental defects may extend up from the cord to the mesencephalon, although they seldom pass up as actual cavities. We shall presently see the importance of this fact.

The symptoms produced by this variable state arise, as I have said, in consequence of the distension of the cavities or the growth of the adjacent tissue. Indeed, some may be due to the absence of the nerve tissue which has not formed. The former generally come on after adult life is reached, but those which are due to simple defect exist from birth. You know the common characteristic of those of the first

class—pain, analgesia without anæsthesia, and muscular wasting when the anterior grey substance in damaged. But the symptoms vary in different cases, just as do the cavities, and they are sometimes such as would not suggest their cause. Indeed, such cavities are sometimes found when no symptoms can be heard of, and we should therefore be prepared for the fact that the symptoms may often be slight, and not characteristic. With unsuggestive symptoms, we can hardly suspect this cause unless there is the additional evidence of imperfect development afforded by defective closure of the spinal bones, called "spina bifida occulta." Such imperfect closure of the bony canal, with no external tumour, affords ground for suspecting a like state of imperfection in the spinal cord.

Let me tell you of a case in which this evidence was most suggestive. A man, thirty-five years of age, had suffered much pain in the arms for eight years. He was a professional musician. Use of the arms in playing relieved the pain for a time and then increased it; the use induced also a feeling of powerlessness. When I saw him the pain extended down the inner side of each arm, and was especially great in the upper part. Objectively, nothing abnormal could be found. There was no tenderness and no deficient sensibility of any kind. The only thing discovered was that the right trapezius was a little smaller than the left, and presented distinctly less excitability and contraction to both faradism and voltaism in all its parts.

The case had baffled the doctors who had seen it; by one it was regarded as myelitis. The key to what I believe was its nature was this fact. At the bottom of the dorsal spine was a peculiar cicatrix, something like the umbilicus, but with furrows running from it a short distance to right and left. On this were a few large hairs, a very suspicious indication. The first two lumbar spines could not be felt; they

Fig. 14.—Position of cicatrix on back.

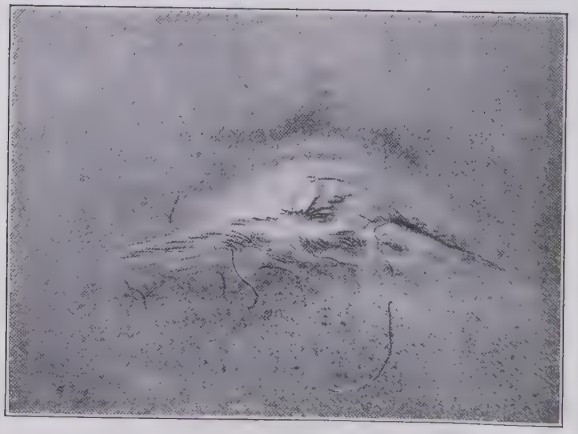


Fig. 15.—Cicatrix, natural size. The large hairs upon it can be clearly seen.

were apparently absent. The scar had been there as long as he could remember, and he had been told that it was present at birth. If so, it seemed to be a case in which an external spina bifida had sloughed away in utero. At any rate it was clearly a case of "spina bifida occulta." This involved a high probability, almost certainty, of a defective spinal cord in that region, and we know how often this is associated with imperfect development higher up, with a fissure or fissures. The fact made syringo-myelia the probable cause of the pain, and we could thus understand why no treatment had had any influence on his sufferings. If the assumed nature of the case is correct, it is an illustration of the various and dubious symptoms which may be produced by such congenital disease.

But it is not with these cases, with trifling symptoms, that I am specially concerned to-day. I wish to direct your attention to some evidence that the existence of these cavities occasionally induces the occurrence of one of the gravest lesions of the spinal cord, hæmorrhage, and determines special, perhaps characteristic, symptoms. I have seen several cases in which the peculiar character of the symptoms can be best understood by ascribing them to a hæmorrhage into such a cavity as we have considered. In some cases, other symptoms made this probable.

First, however, let me give you a fact which proves that this is not a purely hypothetical pathology. I show you sections from a spinal cord in which such syringal hæmorrhage had occurred. A series of the sections are figured in my "Manual of Diseases of the Nervous System" (Vol. I). A cavity passed from the position of the central canal backwards and outwards, along the left posterior horn, and extended from the cervical region to the lower part of the cord. Through part of the dorsal region there was a simi-

lar small cavity on the right side. That on the left was distended with blood throughout. Hæmorrhage into it had apparently commenced in the cervical region, for here the cord was so broken up by extravasation that only a layer of nerve tissue remained outside the blood. It is probable that the blood had escaped into the cavity in this part, and had gravitated downwards, after distending the whole cavity; the resistance caused the blood to work its way into the spinal cord at the seat of the primary hæmorrhage, and to cause death, partly by the local destruction it wrought and partly by the adjacent inflammation it excited, which ascended the cervical enlargement until all the muscles for breathing were rendered powerless.

The case occurred here twenty-five years ago, and the notes are imperfect, but they show certain important facts. The patient was a coachman forty-eight years of age, and, as far as could be learned, he had had no preceding symptoms to suggest the existence of these cavities. The onset was acute, but not instantaneous. One evening, at his work, he found his legs "giving way," but managed to walk home. The next morning he could not stand, and in the course of the day he ceased to be able to move his legs. He did not lose all power of contracting the muscles; this indeed persisted even to the end of life, nearly five weeks after the onset. Such a development of symptoms, acute, but not sudden, suggests myelitis, and so do the facts that the man was not temperate, and had lately been much exposed to cold and wet. But there were other symptoms which indicated more than inflammation. During the first night he had most painful tingling between the knees and ankles, and this was followed by pain behind the knees, and very intense pain in the spine. Even when admitted to the Hospital three weeks later, any movement of the body caused pain in the spine, "as if a knife were driven down it," pain that made him shriek. It

was attended by severe but less sharp pain in the legs, "as if they were being broken across." Such pain does not occur in simple myelitis; it is commonly a result of hæmorrhage. When admitted, three weeks after the onset, he had lessened sensibility to touch over the legs, but that to pain and to heat was increased,—a strange condition, hardly known in myelitis. There was flaccid palsy, with loss of all reflex action. Weakness of the arms developed during the fourth week of the disease, and became complete at the shoulders and elbows, but some movement of the fingers continued to the end. With this loss, movement of the thorax failed on the left side (that of the syringal hæmorrhage) earlier than on the right, and was followed by paralysis of the diaphragm and difficulty in swallowing. During the last days of life occasional contraction of the sterno-mastoid and extra-respiratory muscles alone effected some slight breathing, insufficient to maintain life. He died on the thirty-third day from the onset. Almost to the last he could feebly contract most of the muscles of his legs, and those moving the hands.

I have told you the lesion that was found, extravasation into a cavity beside the left posterior horn, extending from the cervical region downwards. From the chief seat of hæmorrhage some blood had escaped outside the cord. There was no upward hæmorrhage within the cord, but there were signs of ascending myelitis, and to this secondary inflammation we must ascribe the latter fatal ascending paralysis. The precise relation of the sequence of symptoms to the morbid process is not easy to discern, but some important points are distinct. One fact the case illustrates is that the onset of the symptoms of syringal hæmorrhage may be less sudden than those of hæmorrhage usually are. If the blood escapes into a cavity of considerable vertical extent, there is not much erosion of the cord at the seat of the hæmor-

rhage until the cavity is filled. In this case it is probable that such erosion did not occur until after the hæmorrhage into the cavity had ceased, and that a renewal of hæmorrhage was excited by the removal of the patient to the Hospital, and this invaded the substance of the cord at the place of least resistance. In the second place the effect of such hæmorrhage on the function of the cord may be less absolute and less consistent than those produced by a primary extravasation into its substance. Thirdly, the case shows how the effect of such a lesion may be increased by secondary myelitis, a slower subsequent process, the danger of which should always be remembered.

I have mentioned that the opinion has been maintained by some authorities who have given much attention to the subject, that many cavities regarded as congenital are not such, but are the result of hæmorrhage. But the case I have related shows what care is necessary in concluding that a circumscribed space occupied by blood through a considerable extent of the cord, was produced by the extravasation found in it. It might have been thought that, in this case, the hæmorrhage had forced its way down the cord and thus caused the cavity it occupied, were it not for the definite wall the cavity possessed and for the symmetrical fissure on the other side, adjacent to the right posterior horn. It was not continuous with the other, and contained no blood, but the symmetry leaves no doubt of their congenital nature. Were it not for this second fissure, it would have been easy to think that the other was actually produced by the fatal hæmorrhage.

We can understand that hæmorrhage may easily occur into these pre-existing cavities. They are irregular in position and in size, and therefore in their relation to vessels. The adjacent gliomatous tissue, the residual embryonal tissue which surrounds them, seems to be readily broken

down by the fluid they contain. Adjacent vessels are imperfectly supported, and may easily give way. Moreover the gliomatous growth which occurs is vascular and may be a source of hæmorrhage. In a case recently described by Dr. Alexander Bruce,* fatal hæmorrhage had occurred into a gliomatous tumour in the cord, containing cavities. The tumour was apparently due to local growth of residual tissue, but it was so vascular as to deserve the name "angio-glioma." The extent of the resulting extravasation will depend upon the size of the vessel, and, if the blood escapes into a cavity, on the vertical extent of this. Lastly, the symptoms produced will vary according to the force with which the blood flows out, and to the secondary effect of the hæmorrhage. It is important to remember that hæmorrhage may produce its manifestations by destruction of tissue, by pressure, by slower erosion of the adjacent structures, and also by secondary inflammation, such as always results from a traumatic process. Thus, some effects of the condition may not coincide with the primary extravasation, but may be developed as this increases, or they may more slowly follow it.

Let me now mention to you the symptoms of two cases which are, I think, only to be explained by assuming that hæmorrhage occurred into such a pre-existing cavity. It is perhaps significant that the diagnosis has to be a matter of inference and not of demonstration. It signifies that hæmorrhage confined to such cavities is a less fatal lesion than hæmorrhage into the substance of the cord. As we have just seen from the case I have mentioned, the latter may be a secondary effect; hæmorrhage into a cavity may escape beyond it, may work into the substance of the cord, as in the case I have mentioned, with far graver effects than

* Scottish Med. and Surg. Journ., Aug., 1902.

if the extravasation is limited to the cavity, as we may assume it was in the cases I have to describe. They differ widely in their symptoms, but they agree in two features, in the prominence of sensory loss, and in its peculiar seat and limitation, while the first case presents a distribution of motor palsy which it is scarcely possible to explain, except on the supposition I have advanced. In considering the symptoms, remember that the cavities occur chiefly in the posterior region; hence we can understand the degree of sensory impairment. Remember also that a cavity along the posterior cornu if distended by blood, may compress the lateral column, and if it extends forward to the central region, it may compress the anterior horn. Remember, lastly, that such hæmorrhage is very slowly absorbed; ultimately the extravasation lessens in bulk, as the disintegrated blood is removed, and thus symptoms due to compression and not to destructive laceration may ultimately pass away to an extent that would not be expected from their initial degree, and would scarcely be thought possible from the long time during which their severity is maintained. This seems to be the lesson of the first case, and, if it can be trusted, it shows how important it is to recognise the morbid process. It will justify hope, when otherwise there would only be despair.

Case 1.—A member of our own profession, Dr. C., forty years of age, after a good deal of worry and a hard day's work in very cold weather, felt at night a momentary stab of pain in the right side of the head, followed by a peculiar sense of giddiness, objective vertigo in the dark. Although he could see nothing, he felt as if everything before him were passing from one side to the other. Then he fell asleep. At three o'clock he woke up to find his arm powerless. He pinched it and discovered that it was also insensitive. Next morning it was found to be completely paralysed, without

movement and feeling, and so it remained. The condition was just the same when I saw him three months later. Even at first there was no weakness of the face or of the leg. Sudden paralysis of the left arm, preceded by sudden pain in the right side of the head and giddiness, naturally suggested a cerebral lesion, and it was thought by the doctors who saw him that there had been thrombosis of a cortical vessel on the right side of the brain. Such sudden palsy always indicates a vascular lesion, rupture or closure. But there was no heart disease to indicate a source for embolism, and cerebral hæmorrhage at forty without renal disease was most unlikely.

His symptoms, when closely scrutinised, were incompatible with disease of the brain. The points of distinction are instructive. A cerebral lesion never causes complete paralysis of the arm, without any affection of the face or leg. The structures are in such contiguity that absolute destruction of the centre for the arm, or the path for the arm, cannot occur without such implication of the adjacent regions for the face and leg as to cause impairment of their function, at any rate for a time. Moreover, in cerebral paralysis of the arm, the muscles of the shoulder girdle are never completely paralysed. In this case the affection of the muscles was remarkable. There was complete loss of power of the trapezius, of the sterno-mastoid, and of the pectoralis on the left side. Movement of the head to one side may be at first weakened from brain disease, but complete paralysis of the neck muscles never occurs, and the sterno-mastoid is weakened on the side opposite to the affected arm. This peculiar palsy of the neck muscles proved that the condition was due to a lesion of the spinal cord. The affection of sensibility confirmed this in a striking way. There was complete loss of sensibility in the forearm below the elbow, and it was greatly diminished on the outer part of the upper arm and slightly

on the inner side. The greater loss extended over the shoulder and the side of the neck almost to the edge of the jaw, and behind almost to the occipital bone, everywhere ceasing suddenly. It also passed down over the thorax, behind to the level of the angle of the scapula, and in front almost to the edge of the ribs, everywhere stopping at the middle line. On the face, head and abdomen, sensation was normal. Such a distribution could not result from cerebral disease. From this cause the loss which extended up the neck would certainly have passed on to the face and head, and also from the thorax to the abdomen. Thus, this also clearly indicated a lesion of the spinal cord. The suggestiveness of the pain in the right side of the head was lessened by the fact that he had been liable to such pain for some years. Transient vertigo has little localising significance, and, as we shall see, it could be otherwise explained.

The mystery of a spinal lesion so severe, and yet so limited in effect, damaging the left side of the cord so gravely and yet not interfering with the right side, would have been very great but for another symptom, which suggested an explanation for its occurrence and also for its limitation. He had a congenital defect of certain movements of the eyes. They were habitually directed to the left, and could not be moved at all to the right of the middle line. Indeed the left eye was habitually directed outwards mid-way to the outer canthus and could not be brought even up to the mid-position, nor could it be moved outwards beyond its habitual position. Yet it could be moved obliquely downwards and outwards. It could not be moved upwards, and with this was associated a little drooping of the lid. The right eye could be moved inwards, upwards and downwards well, but not outwards at all. Thus there was a congenital absence of movement of both eyes to the right, and of almost all movement of the left eye. Such a condition, life-long, means a congenital defect

of structure in the upper part of the pons on the left side. We know that such defects in the pons may be associated with similar defects in the spinal cord, and that in the cord these take the form of syringomyelic cavities or fissures, usually, as I have said, with an adjacent layer of gliomatous tissue and often with separate tracts of such tissue. Thus, the condition of the eyes revealed the probability of a state of the cord such as might have determined both the occurrence and the limitation of the disease, a congenital cavity into which the hæmorrhage had occurred. A congenital associated defect in the pons would fully explain the peculiar vertigo, since a sudden hæmorrhage into the cord might cause disturbance of the equilibrial centre in that part, especially if the two conditions were structurally connected.

Three months later the loss of sensation had lessened in degree; it still extended over the same regions, although at the margins it passed more gradually into the normal state. The paralysis remained as before; although he could just contract the trapezius and sterno-mastoid, he could not contract the pectoralis. All the muscles had wasted, but only in a moderate degree, and, as at first, there was merely a diminution in electrical irritability, just the same to faradism and to voltaism. We can understand this if the anterior grey matter was compressed by the hæmorrhage but not destroyed. It would be so compressed if a cavity beside the posterior horn extended forwards to the neighbourhood of the central canal. The wrist-jerk was increased and there was some excess of knee-jerk, but without weakness of the leg.

I have not seen Dr. C. since, but I have lately learned that after two years the power gradually began to return, and that he has since regained fair power over all parts of the arm. I am glad to say that absolute confirmation of the diagnosis is still lacking, for he is otherwise in excellent

health. But there has not been wanting some evidence of the accuracy of the diagnosis. You perhaps know that the peculiar joint disease met with in tabes, with enlargement of the ends of the bones, is also met with in syringomyelia. This patient, after the attack of paralysis, had increasing trouble at the hip-joint. He believed that at two years of age the joint had been dislocated and the displacement reduced, but such an accident would manifestly not explain grave and increasing trouble after mid-life. Mr. Symonds took him into Guy's Hospital and operated. He found the head of the femur enlarged to twice its natural size, and he sawed it off. The edges of the acetabulum were thickened to twice the normal. The patient made a good recovery and has since acquired a most useful new joint.

The condition presented so close a resemblance to this special arthropathy as to constitute some corroboration of the diagnosis of syringomyelia, in spite of the absence of any change in the sensation in the leg. As I have said, the great cause of tabes (syphilis) could be quite excluded. But another peculiar symptom occurred, which may be taken for what it is worth. It is said that "phlebitis," *i. e.*, venous thrombosis, is more common in the subjects of syringomyelia than in others. Whatever may be the value of the assertion, this patient had a clot in some veins of the leg on two occasions, first soon after the attack of paralysis, and again after the operation. All things considered, I think we have reason to believe that there occurred, in this case, hæmorrhage into a syringal cavity and that the evidence is as strong as we can expect to meet with, short of pathological proof.

Case 2.—In the next case, also, evidence of a similar lesion is such as to amount to an equally high probability. It is that of a man, the subject of the hæmorrhagic diathesis. From this, the sons of his sisters also suffered, although the

sisters themselves escaped—a peculiarity met with in some other congenital diseases, notably in pseudo-hypertrophic paralysis. At the age of sixteen he gave himself a sudden shock by jumping over a box; he felt little immediate effect, but the next morning had lost power and sensation in the legs. He slowly recovered fair power of movement, but considerable defect of sensibility remained, and was present when I saw him, twenty years after the onset. During the intervening time he had some attacks of transient swelling of the knee-joints, thought to be due to hæmorrhage into them, such as occurs in the subjects of this diathesis. Some defect of power of flexing the ankles was the only motor symptom that continued. He had suffered for years from pains in the legs, intermittent at first, but lately more constant, and he had a troublesome ulcer on one toe, which was obstinate, and when healed was prone to break out again. But the loss of sensibility persisted, and was unchanged when I saw him. It was complete to both touch and pain, and was remarkable in its distribution. It extended over the lower and inner half of each buttock, down the middle of the back of each thigh, the outer part of each leg, and over the feet,—soles and heels, but sparing the inner side, and a small area on the outer side of the left foot had escaped, where there was some hyperæsthesia. With the exception of this hyperæsthetic area, the loss was perfectly symmetrical, and corresponded precisely to the sensory distribution of the last (fifth) lumbar and all the sacral segments of the spinal cord, the distribution of the fourth lumbar escaping entirely, even on the inner side of the lower legs and feet. Such a correspondence to structural arrangement is most unusual in a lesion of the cord such as inflammation or common hæmorrhage; the lesion and its effects have then a random distribution. The peculiar seat can only be explained by assuming a structural condition which determined the effects of the disease.

The only condition we know that will explain it is a congenital cavity or cavities, central, and extending back in the medial line, or bilateral and symmetrical in the posterior columns, conditions which, as we have seen, are occasionally met with. It is easy to understand that the hæmorrhagic diathesis might have caused slow bleeding into such a cavity as the result of the concussion, and a trifling difference in the extent or position of the cavity on each side might have led to the escape of the hyperæsthetic area on the left foot. From what we know of the bleeding in this constitutional condition it is easy to understand that it should have occurred slowly, from a very trifling source, and that thus its effects only became manifest the morning after the concussion. The case agrees with the last, and indeed exceeds it, in the pronounced and persistent character of the sensory loss. Moreover, it agrees also in the presence of another corroborative condition. Joint changes were present here also. There was thickening of the ends of the bones forming the knee-joint, quite like that of tabetic arthropathy, and there were also chronic changes in the hip-joints. Extension of both the knee- and hip-joints was limited by the arthritic changes.

Case 3.—I may mention a third case, which was possibly of the same nature, although the symptoms were less definite. A girl, aged twenty-one, was admitted here with paraplegia of two months' duration. For eighteen months the left leg had occasionally given way for a moment, lately more often, and slight weakness had developed. This sudden transient "giving way" of the legs is often regarded as evidence of hysteria, especially if met with in a girl. It is often, however, a forerunner of organic disease. During the month before the onset of severe palsy, the right leg had shared the weakness of the left, and there had been troublesome cramp-like

pains in the feet. Seven weeks before admission complete paraplegia came on very rapidly. The legs one day were much weaker, and the next were completely paralysed in motion and sensation, with much pain in the left leg, and for a day or so acute pain in the spine. A pain around the abdomen at the level of the umbilicus was felt for a few hours from time to time. On admission, the ankles could be slightly flexed and all the intrinsic muscles of the feet could be contracted. The calf muscles were powerless, and so were those of the knee- and hip-joints. The knee-jerks were lost, and there was no plantar, abdominal, or gluteal reflex. No faradic irritability could be elicited in the glutei, the flexors of the knee, or the calf muscles, but it remained in the extensors of the knee, better on the right side, and it was present also in the peronei and tibialis anticus of the left leg. Voltaism seemed to be lessened in the same way as faradism. The muscles were not much wasted, and I may anticipate by saying that during her stay in the hospital (four months) the muscles gradually recovered, and all regained good faradic irritability. Their perfect recovery thus makes it improbable that there had been a primary lesion of the anterior horns, such as acute poliomyelitis, a fact which renders the sensory condition more important.

Sensation to touch was lost over both legs, except on the inner side of the right leg above the ankle, but it was absolutely lost on the corresponding region of the left. It was lost on both soles, including the heels, and this loss extended over the outer half of the dorsum of each foot. But this plantar loss on the left foot spared the last phalanges of the toes, while on the right foot only those of the great and little toes had escaped. On the lower legs, loss was not quite absolute below but became so about three inches from the knee, and this continued up the back of the thighs and

over the gluteal region to the crest of the iliac bone, where it was succeeded by a zone of hyperæsthesia, extending up to the ribs. It was absolute also on the front of the thighs and lower part of the abdomen, in the hypogastric and inguinal regions, becoming less in the umbilical region, but only normal near the ensiform cartilage. The alteration to pain corresponded in general with that to touch, but on the soles, where touch was lost, pain was only delayed, and so also on the back of the left foot. There was the same increased sensitiveness to pain as to touch on the back, above the iliac crest.

A month after admission she could perform all movements of the legs, but sensation was almost the same. It slowly improved, by general diminution rather than by local change. When she left the hospital there was only absolute loss in the hypogastric region and on a spot on the outer side of the right foot, and on the inner side of the left leg. The knee-jerks were still absent; the legs could be moved freely and with force in any way as she lay, but it was impossible for her to stand on account of the extreme degree of incoordination.

I think that this case may possibly have been syringal hæmorrhage. It is certainly one of disease causing extensive abolition of the functions of the lower part of the cord, and the predominant change in sensation, its persistent character, the enduring loss of the knee-jerk, and the extreme residual incoordination, all indicate damage to the posterior columns, apparently interfering with the posterior nerve roots before they reach the grey substance. The degree and general symmetry of this damage suggest hæmorrhage into a cavity or cavities.

I have seen some other cases which I think may be of the same nature, but they are not sufficiently pointed in their indication or precise in their record to make it worth while to take up your time with their details. Those which I

have mentioned are sufficiently suggestive to warrant the pathological conception of hæmorrhage into pre-existing cavities, "syringal hæmorrhage." As I have said, we may reasonably assume that extravasation may occur into any cavity, irrespective of its origin. The vascularity of adjacent gliomatous tissue, especially when this takes on a process of growth, is often great. It is intelligible that these should sometimes give rise to hæmorrhage, and it is therefore not surprising that such growths should sometimes contain the remains of small old extravasations. Little significance can be ascribed to these as regards the original causation of the cavities with which the condition is associated. I refer to this incidentally. My chief object is to call attention to the class of symptoms which seem to indicate hæmorrhage into the cavities. I hope that further similar facts may be ascertained. For actual confirmation we may have to wait. As I have said, purely syringal hæmorrhage seems to be seldom fatal and the fact makes the diagnostic question of great importance. Death seems generally the result of the eroding extension of the extravasation into the substance of the cord, or a later result of secondary spreading inflammation, which is so grave when excited by an irritant cause such as hæmorrhage.

If any therapeutic lesson is to be obtained from the cases I have described, it is the extreme importance of rest, perfect and prolonged, in every case of sudden spinal palsy, especially when pain suggests a hæmorrhagic cause. If removal to a hospital is necessary, the patient should be completely passive. The slightest effort on his part to assist may increase the hæmorrhage, or start afresh that which had ceased. Moreover, not only should everything be avoided that may increase the blood pressure in the spinal cord, but an endeavor should be made to lessen that which gravitation tends to cause. If possible, the position should be such that the spine is not the lowest part. There is a common

tendency in arranging the treatment of affections of the spinal cord, to consider the organ more than the process. In acute affections, at any rate, this is a mistake. It may be said, as a general rule, that in proportion to the acuteness of disease, the elements of treatment should be determined by the nature of the morbid process rather than by the nature of the organ affected. The importance of this is clear if you consider the lesion we have been discussing, hæmorrhage. The organ into which it occurs is of slight importance compared with the nature of the lesion, so far as treatment is concerned. The object to be kept in view is to promote the cessation of the escape of blood, and for this no influence is too slight to be neglected, and the effect of gravitation, for good or ill, is often overlooked. In spinal hæmorrhage, whenever it can be endured, the prone position of the body should be maintained; and if this is impossible, the patient must be kept on the side. If hæmorrhage occur into the leg the limb is not allowed to hang down, and if it is even suspected in the spinal cord, this should not be the lowest part of the body, as it is when the patient lies on the back. Indeed, I think that, in some cases at least, the sitting posture, with legs depending, would be the most likely to promote cessation of the hæmorrhage, if only perfect stillness can be maintained. But remember the importance of posture in all acute affections. If you only carry away this lesson you will not have listened in vain, for it is one that you are sure to need in your future work. You may never meet with a case presenting such symptoms as suggest syringal hæmorrhage, but you are sure to meet with cases of acute disease of the spinal cord. You will be able to understand them better, and deal with them more wisely, for your study of such cases as I have described. You cannot gain real knowledge, of any kind, that has not far wider applications than is at first apparent, or may not help you when and where you least expect.

LECTURE IX.

MYASTHENIA AND OPHTHALMOPLEGIA.*

The mysterious malady commonly called "myasthenia," or "myasthenia gravis," is a rare disease, the special characters of which have only been discerned during recent years. Indeed, it is still not known, even by name, to many members of the profession. It is met with chiefly in the first half of adult life, and is characterized by general feebleness of the muscles, and also by their quick exhaustion on use, and the quick renewal by rest of what power they possess. The same feature is often, but is not always, conspicuous when the muscles are stimulated by electricity; the effect of a tetanizing faradic current of given strength soon, as a rule, ceases, but returns after a brief rest. This weakness is not attended by definite wasting, or loss of electrical excitability. Although general, it is especially marked in the lips, palate, pharynx, and often in the muscles of mastication, and in those of the eyeball. The "bulbar" weakness, indeed, first attracted notice, and the condition has hence been termed also "myasthenia bulbaris."

The course of the disease presents curious variations, but is seldom definitely progressive, nor has great improvement

* This article is not, strictly speaking, a lecture. It was contributed to the special issue of the "Deutsche med. Wochenschrift," in honour of the seventieth birthday of Professor v. Leyden. An English version, rewritten, appeared in the "British Med. Journal," May 25, 1902.

been often seen, except as a transient event. Death has been the result of intercurrent disease, and has yielded no clear indication of the nature or even the seat of the malady. In the absence of fact, theories have abounded. Treatment has seldom had marked effect.

The symptoms vary in different cases, and in some there has been considerable loss of power in the eye muscles. The object of this paper is to describe three cases in which this feature was very marked, and also to call attention to another symptom which each presented, a peculiar alteration in the smile, due to the absence of the normal action of the zygomatic and risorius muscles. The general features of the disease will be perceived from the account of the cases; the special symptoms can be afterwards described and an attempt made to discern what indications they suggest regarding the nature of the disease.

Case I.—This patient came under my observation long before myasthenia became a clinical conception. She was a girl of twenty-three, and was seen first in 1874, and from time to time during the following four years. Soon after this she died from some intercurrent disease. My notes are incomplete, but they record the chief features of the case.

No family history of neuroses could be ascertained, no preceding disease, nor any exciting cause for the affection. Beyond a tendency to headache she had been well until the age of twenty-one, when her symptoms were first noticed; they commenced gradually, and slowly increased. During the next six months they increased slightly, but afterwards were almost stationary, some indeed diminishing. The aspect of the patient at once attracted notice by the stiff look of the eyes, and the unnatural expression of the lower part of the face. On closer observation, it was seen that the latter depended on the absence of any natural smile. Instead of a movement outward of the angle of the mouth, when she smiled

or laughed, there was only an elevation of the upper lip. There was no action of the zygomatics or risorius, but the levators raised the upper lip, causing a furrow above it beside the nose, and even wrinkling the skin there. Thus there was only an unpleasant snarl as the expression of a pleasant emotion. There was slight drooping of the eyelids, greater in the left. Movement of the eyes was much lessened. That of the left eye, outwards and inwards, was reduced to one-eighth of an inch. The right could be moved inwards to the full extent, but not outwards beyond the mid-position, to which it was brought back. Hence an attempt to converge caused an excessive movement of the right eye. Both moved downwards freely; upwards there was a very slight movement of the right eye above the mid-position, none in the left. The fundi were normal, and so was vision. The pupils reacted to light. She complained of difficulty with the lips, but could articulate fairly. She could not narrow the mouth to whistle as she formerly could. The voice was nasal. The arms were weak, the grasp with the dynamometer being only 10 kg. each. Extension of the fingers was the only movement distinctly deficient. They could not be straightened if the wrist was over-extended. Her legs were readily tired, but she could walk a mile. Her knee-jerks were afterwards found to be normal. There was little change in four years during which she was occasionally seen. Then she died, from what cause is unknown.

Case II.—A girl, aged twenty-nine, was sent to me on account of general muscular feebleness, which was first noticed three years before, at twenty-six. The feebleness had come on gradually, in arms and legs, without change in sensation or affection of the sphincters. Her aspect at once attracted notice. There was double ptosis, so that the lids concealed a third of the cornea, but the lids were sometimes fully raised. The eyes could not be moved upwards, and

very little downwards, the right eye could only be moved outwards to half the normal distance, the left eye still less. Convergence was lessened, and there was slight nystagmus on looking to the left. The pupils acted to light, and slightly on convergence, but accommodation was normal. The eyelids could be closed, but only feebly. The smile consisted only in elevation of the upper lip, the zygomatic muscles being apparently inactive, but a slight furrow occurred on the left side, perhaps from the risorius. The lips were weak, and she had lost her old ability of whistling. The voice was somewhat nasal, and the palate was but little raised on phonation. When the patient was tired, swallowing was difficult, and liquids occasionally regurgitated through the nose. The muscles of mastication were so readily fatigued that the patient had often to rest two or three times during a meal. The muscles of the neck were weak, and the head was kept upright with difficulty, tending to fall backwards or forwards. The arms were feeble, and if used quickly, became still more so. The legs were also weak, and she could only walk two or three hundred yards. A movement requiring force, such as flexion of the hip, could not be sustained for more than a few seconds. The knee-jerks were normal, and there was no foot clonus. There was no wasting. The electrical irritability of the muscles was normal and so was sensation.

Besides nourishing food, she was treated with gentle massage, tonics, and the hypodermic injection of strychnine. The result has been a slight but distinct improvement during the three years up to the present time. I have seen her at intervals of about six months, and without giving the details of her variations, I may describe her present state as observed in March, 1902.

The patient's general condition was the same, but the improvement in many symptoms was conspicuous. The

smile, levator only, was little changed except that on the left side, in laughing, there was the same slight outward movement of the angle of the mouth. The naso-labial furrow, on smiling, still extended from the nose to just above the corner of the mouth (see Fig. 16). The lips were distinctly stronger; although she could not whistle even a year ago, she had done so on several occasions, loudly enough to call a dog. The tongue was protruded well, and the palate was raised a little better than nine months before. Fluids had ceased to regurgitate through the nose, the voice had lost its nasal character, and choking on swallowing had quite ceased. The masseters contracted strongly, but were soon tired, and the patient had still to rest from eating several times in the course of a meal. The ptosis continued and the eyelids could not be raised higher by the will, although still when she was interested, they were raised excessively, enough to show the sclerotic above the cornea. This was not attended by any overaction of the frontales, but the patient could contract these slightly by an effort of the will. The orbicularis could close the eyelids, but still with little force; the contraction could be easily overcome. The movements of the eyes were as follows: Upward movement was absent in each; the downward movement was only a little less than normal (eighteen months before it was very slight). The right eye moved outwards (external rectus) about three-quarters of the normal distance, and the associated movement of the left eye (internal rectus) was about half the normal. The two eyes were moved to the left only about one-sixth of the normal, the same degree in each. The lateral movement of each eye separately was the same as when tested together. The lateral movements were attended with a curious disposition of the eyes to move downwards, greater when tested separately than together. No nystagmus could now be observed, but on looking to the left, the left

upper eyelid presented up and down nystagmoid movements. (I first noted this two years before in the same degree.) Convergence was weak; for an object 6 in. away the movement was only half the normal; it did not increase when the object was brought nearer, and soon diminished, the right eyeball first moving outwards. The pupils reacted well to light and on convergence. Accommodation was fair, but seemed to be sooner exhausted in the right eye than in the left; there was slight hypermetropia. No weakness in the neck could now be observed, conspicuous as it was at first. The fingers could be completely extended, but the grasp was weak, although only slight evidence of exhaustion could be ascertained, successive efforts with the dynamometer resulting in a pressure of 15, 16, 17, 21, 14, 12, 13, and 14 kg. She had become able to write a long letter without fatigue, which was impossible two years before. In the legs, flexion of the hip was difficult; when sitting, the patient could only keep the foot a few inches from the ground for two or three seconds. The flexors of the knee had fair power, although less than the extensors. The knee-jerks were active, and six repetitions caused no diminution. It was not practicable to test thoroughly the "myasthenic reaction," but the application of a tetanizing current to the extensors of the fingers for about a minute caused little indication of diminished irritability. In the zygomatic muscles, no contraction could be obtained on the left side, but only a slight trace on the right, and this may possibly have been due to stimulation of the risorius. The patient presented nowhere any local muscular atrophy, either in the trunk or limbs. She was thin, her weight being 98 lb. and height 5 ft. 6 in.—about two-thirds of the average weight for her height.

These two cases present a striking resemblance, being of the same sex, not far apart in age, and alike in general and local symptoms, and in the course of these. Muscular

exhaustibility was not noted in the first case, probably because it was not looked for.

Case III.—The last case differs from the others in the later age of the patient, thirty-nine (the symptom dating from thirty-seven), and the fact that the defective power in the limbs was much less prominent. Nevertheless, the resemblance in the slight bulbar symptoms, in the condition of the face, and the defective movement of the eyes was so close as to show that it was of the same nature. It is not, however, worth while to describe it in detail since the account would be chiefly a repetition of the symptoms of the last case. The special features will be mentioned in the discussion of the particular symptoms.

Remarks.

I do not propose to discuss the general features of the disease. The question of its nature will be presently considered, and the relation to it of the symptoms to which I desire especially to direct attention. These are the peculiarity of the lower facial movements in emotional expression, and the defect in the movement of the eyes.

The *nasal smile* is, perhaps, the most convenient designation for the peculiar character of the smile presented by each of these patients, although it might also be called the "levator smile." Its feature is the absence of the normal movement at the corner of the mouth, which either carries the furrow from the nose around the corner of the mouth or produces a separate depression there. In these patients the furrow of the smile was entirely above the upper lip, ceasing outwards above the angle of the mouth. It is less marked in Case II than in either of the others, although I am only able to give an illustration of this case (Fig. 16). Its abnormal

character may not be so conspicuous at first as when it is observed more closely, and especially when it is compared with the normal smile in a person of the same general aspect. Fig. 17 is from a photograph, which has been kindly taken for me by Dr. Farquhar Buzzard, of the effect of gentle faradic stimulation of the levator labii superioris in a patient

Fig. 16.—From a photograph by Dr. Batterbury, of Berkham sted, of the patient Case II, showing the limitation of the smile to elevation of the upper lip and the absence of move ment outwards of the angle of the mouth. The photograph gives only an imperfect indication of this characteristic symptom.

Fig. 17.—From a photograph by Dr. Farquhar Buzzard, showing the similar furrow caused by isolated stimulation of the levator labii superioris.

whose face was anæsthetic in consequence of the removal of the Gasserian ganglion by Krause's operation. The slight contraction of the orbicularis palpebrarum is due to the fact that some of the branches to this muscle cannot be avoided. It will be seen that the furrow produced corresponds in position to that of the patient's smile. In Cases I and III the

limitation of the contraction to the muscles raising the upper lip was even more marked. In the case figured, on an energetic laugh, there was a slight movement on the left side of the mouth, probably by the risorius, which only made more conspicuous the absence of the normal movement.

The fact that this nasal smile was so conspicuous in each of the cases raises the question whether it may not be a common feature of the disease. It does not seem to have attracted attention, but it might easily escape notice. In several recorded cases, "weakness of the lower facial muscles" is mentioned, without being definitely specified.*

The Ophthalmoplegia.—Loss of power in the eyeball muscles has been mentioned as among the symptoms of many cases of myasthenia, but unfortunately has seldom been carefully described. In these three cases it was a most conspicuous and enduring symptom. It presents, at first sight, a strong resemblance to the ophthalmoplegia from nuclear degeneration. Yet there are some noteworthy differences, possibly of much significance. One of these is the greater escape of the muscles moving the eyes downwards, and the implication in various degree of those moving the eyeballs upwards. Not less striking is the constant and irregular affection of the lateral muscles, the fact that this was different in degree in associated muscles, and that in some muscles it

* This nasal smile or nasal snarl, as it might be called in its extreme form, bears a curious resemblance to the movement of the face by which most monkeys express pleasure. This is well seen in the figures in Darwin's "Expression of the Emotions." It raised the question whether animals possess zygomatic muscles. By the kindness of Mr. Beddard, of the Zoological Gardens, Dr. Finny has made some dissections for me, which show that the zygomatic muscles are present in the animals, although they have not quite the same form as in man. How far their inaction in expression is the result of a difference in their direction, consequent on the different shape of the face, is a question still to be decided.

presented extraordinary variations at different periods. In order to present better these features, I have brought together the conditions in the three cases in a diagrammatic form which will be readily understood. Each outer circle represents the normal extent of movement of the eye, and the length of the lines indicates the proportion which the actual movement bore to the normal. Where two or three lines are together they represent the variations of power at different times.

In all cases the light-reflex of the iris was perfect, and I believe that this has been the case in all recorded cases. The condition of accommodation was not easy to ascertain, on account of the presence of hypermetropia, but in two it seemed impaired, and the action of the pupil on accommodation seemed impaired with it, but the hindrance to convergence made the observations on this point uncertain.

Ptosis was a marked feature, as in most cases, but there was no correspondence between its degree and the impairment of the upward movement of the eyes. Equally common was the weakness of the orbicularis palpebrarum, which was so marked in the second and third cases, while in the second case the weakness of the frontales was also conspicuous.

My chief object is to call attention to the peculiar effect of the facial weakness, in altering the smile, and to the peculiar characters of the ophthalmoplegia, in the hope that future observers may ascertain how far these are common. I do not propose to discuss the pathology of the disease generally, but I may point out the possible bearing of the facts on the nature of the malady, a subject which remains altogether mysterious.

The pathological problem is whether the symptoms depend upon a change in the muscles or in the nervous system,

or in both. The "nasal smile" is only one feature of the distribution of the weakness of the facial muscles. The

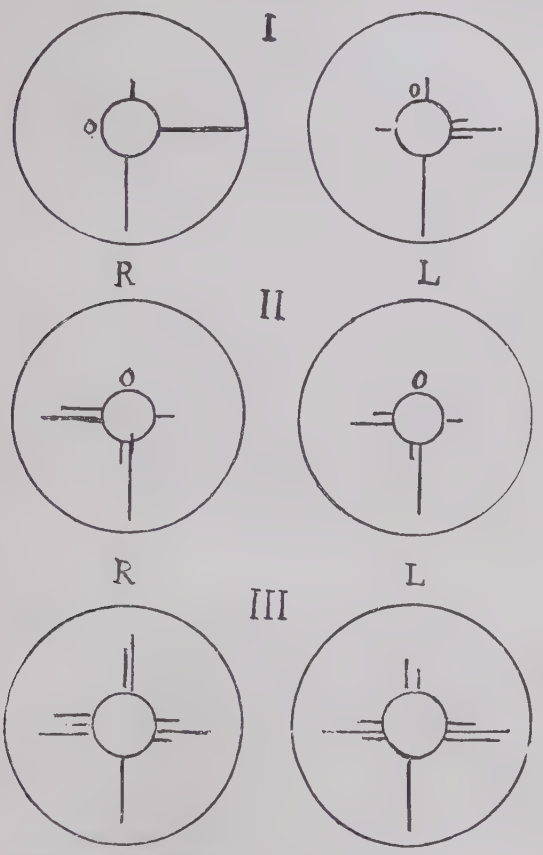


Fig. 18.—Diagrams to indicate the amount of movement of the eyes, R and L, by the recti, in Cases I, II, and III. The outer circle represents roughly the normal extent of movement in each direction. The lines of different length placed together indicate the variations in the degree of the same movement at different periods of the case.

significant fact regarding this is that it corresponds more closely to that met with in idiopathic muscular atrophy (mus-

cular dystrophy) than to known affections of the nervous system. This correspondence (which has been insisted on by Toby Cohn) is seen in the weakness of the frontales, the orbiculares palpebrarum and of the zygomatic muscles. The resemblance is strikingly shown by a comparison of the facial symptoms of myasthenia with the condition of the face in primary myopathy.* The latter, however long its duration, does not involve of necessity any secondary changes in the motor nerves. Slight changes might, indeed, have been expected from the absence of function, but they are apparently not such as to be recognizable by any method of investigation. It is important to call attention to this fact, and the absolute weight of evidence of it presented by Spiller's case (among others), because it is very rare for a case to be so carefully observed during so long a period, and to become the subject of such careful pathological research. The facts also show how much caution is needed before assuming a simultaneous failure of vitality on the part both of the muscles and the motor nerves.

A degeneration of the nerves involves also that of the muscles, and the coincidence of the two should only suggest that both are primary, when there is conclusive proof of truly simultaneous affection, which is not easily obtained.

In myasthenia, the distribution of the symptoms in the muscles supplied from the medulla oblongata also corre-

* Compare the facts mentioned in the lecture on Myopathy (p. 121). A very similar paralysis was present in the case figured in my "Manual," 3d edition, vol. i, p. 589, which is certainly a case of muscular dystrophy of the facial type (the type called that of Landouzy-Déjerine). It is equally striking in the remarkable case of this type which was described by Duchenne in 1872, and also by Landouzy and Déjerine in 1886, and of which the pathological condition has only recently been described by Spiller. The frontales and orbiculares were entirely degenerated, and the zygomatic muscles almost entirely. Complete as was the degeneration of a large number of the muscles, the motor nerves were absolutely normal.

sponds to that in muscular dystrophy rather than in known central affections. A striking feature is the escape of the tongue and larynx, and the moderate degree of weakness of the lips and palate. In the cases I have here described, the variation in the degree of this weakness at different periods was as marked as was that of the eyeball muscles. The absence of perfect bilateral symmetry was as marked in the masseters, in one case, as in the eyeball muscles.

But the ophthalmoplegia, from its resemblance to the nuclear form, may be regarded as suggestive of central disease. It is important, therefore, to note the special features to which I have called attention. Future careful observations may increase or diminish their significance. In its irregular distribution and variations, it presents some correspondence to the weakness in the muscles supplied from the medulla. It is, moreover, important to remember that the ocular muscles are part of the general muscular system. We are prone to associate them with the globes; they move in a special relation, but we know no reason why they should not suffer in a widespread muscular malady.

The information afforded by the microscope regarding the nature of the disease is purely negative. The latest methods of research disclose no structural change. This can surprise no one who has considered carefully the features of the malady. Where there is the capacity for normal action, although in diminished degree, normal structure would be anticipated, and an undue quickness of exhaustion would not be expected to be accompanied by visible alterations. The ability for sustained exertion depends on the capacity for immediate renewal of the elements lost in function, a quick renewal by vital metabolism. The exhaustibility in myasthenia must depend upon defective capacity for this prompt vital nutrition, on a too tardy renewal of the molecules to permit exertion to be sustained. When we remember how

far below discernment, or any promise of future discernment, the molecules of living tissue are, it will be realized how vast may be the imperfection of their nutrition, which can only be revealed by its functional effects. Hence we are only likely to obtain any pathological facts regarding the seat of the disease when local changes proceed far beyond the characteristic functional impairment and involve atrophic degeneration. Atrophy of a few muscles has been noted in rare cases, irregular in distribution, but as yet they have afforded no reasons for regarding the malady as other than muscular.

If present facts only enable us to regard myasthenia as a nutritional disorder, impairing the vital processes in the muscle, we should remember that this is hardly more than a statement of the obvious symptoms in pathological terms. It is only more by its implied negation of other morbid processes. The cause of this nutritional incapacity is, so far as we can yet see, the great problem of the disease, and it is the same, whether the malady is regarded as muscular or neural. Moreover, we must remember that nutritional disease differs from organic degenerative disease only in degree. It consists in changes too fine to be discerned. It may differ in its capacity for recovery, but not in all cases, and not, usually, in myasthenia.

The theory has been put forward and advocated that the impairment of function and vital nutrition can be best explained by ascribing it to a toxic agent. The theory is indefinite, but is apparently based on the mysterious selective action of toxins, on the symmetry of their influence, and on the theory that the effects of fatigue are due to the production in the muscles of a toxic material. But when full weight is given to all these facts, their amount is very small. The argument is purely one of analogy, a proverbial pitfall in reasoning. All that it amounts to is that toxic influences may cause symptoms resembling those of myasthenia in some super-

ficial characters. No one has yet suggested that muscular dystrophy has this origin, although it resembles myasthenia in some important features, among them in the symmetry on which so much weight is laid. The symmetry of the action of toxic agents depends on nutritional susceptibility, and proves no more than this. It is very difficult for any one who has watched cases of myasthenia during years to conceive that the condition is due to a toxic agent, in the absence of any conceivable source for such an influence. The persistence of the symptoms, their slight inexplicable variations in detail and degree, make it difficult, at present, to go beyond the obvious fact of an imperfection in the vital nutritional power, which seems to be a defect of life, with little tendency to progress or improvement. We know, indeed, that many morbid states which have like manifestations may be brought about by divers causes, but where facts are few the need is all the greater for careful scrutiny of the evidence on which theories are ostensibly based.

In recorded cases little benefit has seemed to result from any method of treatment. But in Cases II and III here described the effect of the hypodermic injection of strychnine seemed distinct. Especially in Case II its influence has been marked and sustained, and, in the opinion of the patient, her friends, and her immediate medical adviser, admits of no doubt. In another case, quite similar in general features, its use has been attended by even greater improvement, which seems enduring.

LECTURE X.

THE USE OF DRUGS.

Delivered before the Harlesdon Medical Society on October 11, 1895.

Mr. President and Gentlemen:—I am glad to have an opportunity of addressing members of a branch of the profession to which I have not the honour to belong, a Society of General Practitioners. The chief line of cleavage which runs through the profession is that which separates the many, whose work is primarily to apply knowledge, and the few, whose work it is primarily to advance knowledge, and also to apply, when needed, such particular knowledge as their work has enabled them to obtain. I use the word "honour" advisedly, because I hold that of the two branches, the general practitioner and the physician, the work of the general practitioner is the more deserving of honour. His work is, perhaps not more arduous, and perhaps, on the whole, has a more adequate practical reward, but it is destitute of the incentive which science supplies to those who serve her with pure heart and earnest endeavour, and which makes the labour, at least for such, its own reward. I have also used advisedly the word "particular" in contrast to the word "general." The accurate antithesis would be "special." But this word now involves other conceptions. I think, indeed, that those who are strong in their objection to specialism in medicine scarcely realise how inevitable specialism is. It begins as soon as the boundary

of general practice is passed. The separation of medicine from surgery is itself specialism. But on this subject I need not dwell. I need not emphasise the necessity of restriction of range of work in order to secure depth, or the mistake that is made when specialism is confounded with exclusivism. Yet I must, in passing, ask you never to forget the important fact that the special implies the general, and that no man can be a true "specialist" who has not an adequate practical aquaintance with the whole range of at least one of the two great branches of professional knowledge. Every province of disease abounds in illustrations of the degree to which the special involves the general. In the case of sudden lesions of the brain, hæmorrhage or softening, the treatment needed is only in a small degree treatment of the nervous system. For the most part, it is treatment of the vascular system, or of the heart, or of the general blood state; and any good that can be done is to be effected only through a knowledge of all these morbid states and of their treatment. As a matter of fact, diseases of the nervous system, so called, are widening out into other provinces in ever-increasing degree. The extent to which symptoms directly of nervous nature depend upon blood states and constitutional conditions is becoming more and more apparent, and compels, not only wider knowledge, but greater familiarity with such diathetic states and their indications, many of which are only disclosed by their effect upon the nervous system. There is no part of medicine in which a so-called "nerve specialist" must not be at home. No part of pathology is, or can be, isolated. Unless special work is based upon general and thorough knowledge, it is insecure, and those who trust to it are unsafe These facts should be recognised by practitioners. Consultants are, for the most part, that which practitioners, by their action, decide.

It may occur to you that, in speaking of the general practitioner, I am speaking of that of which I know little. But I count myself fortunate in having entered the profession by the path which so few now tread—that of apprenticeship to a country practitioner. The changing times, the increased demands of the curriculum, and the long period of formal work, have made this method rare. Perhaps the change is wise. The first two years are now spent differently. And yet I learnt much which I would not have willingly foregone during the period in which my professional work consisted chiefly in dispensing medicines or assisting at a small operation. Summer afternoon rambles enabled me to do that which I have since found most useful —to learn much of practical British botany. Botany stands low among the subjects of medical study in professional estimation. But I am certain that there is no part of science which is indirectly of so much service as practical botany. The process of the identification of plants by the descriptions, the training it involves in accurate observation, in careful comparison, and in giving the proper relative weight to different features, is essentially the same as that which is needed in the diagnosis of disease. No subject affords mental training quite so effective for the practitioner's work.

I also gained two perceptions of the work of the practitioner which I have never lost. The first is the narrow limit which circumstance sets to the therapeutical scope of the average practitioner. By "average practitioner" I mean the practitioner whose work is that of the majority, the work which lies among the lower classes, or among the lower middle classes. If the work of practitioners of this country were measured by statistics, I believe these classes would make up four-fifths of it. Of such work, is not the following statement true? With the exception of the cases of acute disease, in which the means are more varied by which the

sufferer may be kept in life and be conducted back to health, the therapeutical means the practitioner can employ are almost limited to the administration of drugs. His means are almost restricted to the method of treatment to which it is now customary to refer in terms not far removed from those of contempt. Where is the room in such practice for the many measures of which we hear so much in the present day—massage and the like—measures which are developed in such elaboration of detail, and described in even a greater elaboration of terms? These systems of treatment, of more or less utility, pass in waves of fashion across the surface of suffering humanity with well-filled pockets. But they are not within the repertory of practitioners whose lot is cast in places less happy, and whose patients belong to classes less favoured.

The second fact that was impressed upon me was that this method of treatment by drugs does, in a large number of cases, not only a definite but a great amount of good. This impression was derived from observation of the effects of such treatment in cases where observation is free from the complication of other influences, a source of fallacy inevitable when patients are admitted to a hospital. When the administration of medicine was the only new element introduced into the problem, the good observed to follow may securely be assigned to it. This condition is often obtained in the treatment of hospital out-patients, and sometimes in the case of private patients. I believe that those practitioners who closely watch the effects which follow, or seem to follow, the measures they adopt, will agree with me that the method of treatment to which the average practitioner is chiefly restricted has not only a power that can be realised now, but also a potentiality yet to be developed.

It has, therefore, been with great interest that I have seemed to see of late more clearly why this should be. The

scepticism regarding the use of drugs to which I have referred, is, I think, less now than a few years ago. One favourite foothold was the statistical demonstration of inutility, as, for instance, that the average duration of acute rheumatism was the same whatever treatment was employed or when no agents were used. Numbers must be great indeed to eliminate fallacy, and even statistics have failed to demonstrate the inutility of drugs, since help has been sought from the willow which casts its shadow on the damp places in which some forms of the malady lurk. We cannot doubt that that which has become true of one disease, definite control by a fresh agent, will be true of others. The empirical advice, "Try all things; hold fast to that which is good," to which, in the hands of peasants, we owe many of our most precious aids, has been applied to the results of modern chemistry, and many new products of science have been seized by the "advanced pharmacy" of the present day, have been tried and found useful, although not often in the diseases in which they were expected to do good.

But can we discern the reason why drugs are useful? Can we not, at least, discern the nature of the reason? Here is a fact, the meaning of which is of far-reaching significance. I show you two tubes. Each contains a small quantity of a white powder—about half a teaspoonful. Each powder consists of the same elements, oxygen, hydrogen, nitrogen, and carbon. One is practically harmless; the other contains within it the power of death to 1000 men. The one is quinine; the other aconitia—the alkaloid which makes so deadly the plant whose flower our ancestors called "monkshood," in the far-off days when the original was often before their eyes. It is an almost startling fact that in this minute quantity of powder, hardly visible to those at a distance, there is such a potentiality of death. Picture to yourselves 1000 men. That which is in this tube would end the life of

every one of them. Here is a latent power beside which the lightning flash is feeble, and to which the earthquake might give place, so far as the comparison depends on lethal certainty.

But the resemblance in the aspect of these two substances is not all. As I said, each consists of the same elements—each is made up of carbon, nitrogen, oxygen, and hydrogen. Each consists of the elements which compose air and water, with carbon added. Why is one almost harmless, and the other a most deadly poison? I might ask the question regarding many other substances composed of the same elements, but between these two the resemblance is strikingly close. The answer to my question may be given, "It depends upon the chemical constitution." True, but this takes us a very little way. When we discern that the difference depends upon the way in which the elements are arranged in molecules and the molecules are grouped together, we are not much nearer an explanation. We see a little more, however, when we realise that chemical constitution means that energy is held "latent" (as it is said), ready to be released when the elements form simpler, closer, compounds. All vital function of the body depends on a like simpler closer union of the elements which make up complex organic compounds. As far as we can see, all the energy which is released in the animal body is released in consequence of chemical action under the mysterious influence of life. Where such closer union of the elements and such release of latent energy are going on, the process may be changed entirely by the contact of molecules of allied constitution, with latent energy on the point of release, so held as to blend with that which is being set free in the living tissue. Blending with this, it may augment or oppose it. Remember that difference in chemical constitution means difference in the readiness with which the elements separate

and reunite, and release their energy. Remember also that minute differences in constitution enable these chemical compounds then to blend with the vital action in one structure, or to be absolutely inert. It must depend on differences in the vital chemistry which underlies function, although these differences which determine affinity or indifference we can discern only by the result.*

Nerve force, as far as we can see, is the result of chemical changes occurring under the influence of life in the molecules which compose nerve tissue. Chemical processes, the breaking up of complex compounds and the formation of simpler compounds, with consequent release of the energy held latent in the former, is the constant element in the production and conduction of nerve impulses. Some chemical compounds may come into relation with the tissue in which the change is occurring without exerting the slightest influence upon it. But another substance may come, even in amount inconceivably minute, whose molecules are so arranged as to fit in, as it were, with the changing molecules of the living tissue. The energy the new molecules bear seems to blend with that which is in process of ordered release in the living tissue, and to blend so effectively as to derange it entirely. The various nerve tissues which compose the centres seem to us the same, but they must differ in their precise chemical nature, and in the precise chemical action which their life-work involves. This fact is revealed to us only by their response to different chemical agents, and the result must be due to a difference in that on which the agent acts. Although we have no other evidence of this, consider the absolute significance of such facts as that atropine acts first upon the nerve substance of the eye, and strychnia upon that of the

* The subject is considered in detail in "Dynamics of Life." London, Churchill's, 1894.

spinal cord. Such an influence as I have spoken of seems to be exerted widely in the case of aconitia. Its contact with some acting nerve structures seems to be so instant and precise as to induce the production of an excess of energy, sweeping all before it; on others, to oppose the process, to induce a sudden stillness among the changing molecules, and to arrest all action. Among the nerves thus influenced may be those on which depends the action of the heart, and with a sudden spasm or a sudden stillness, the heart stops and life is ended. That which so acts we call a poison. Widely different from this is that vehicle for energy which we call a food, yet the difference is one of degree. Every form of food is such by taking to the tissues new energy in fresh molecules. Between the two—the poison and the food—there is an almost infinite gradation of substances whose influence is exerted in the same way, by chemical compounds upon the chemical processes of life as well as on the state of nutrition, and on the mode of action of the living tissues. Between the two there is a like gradation, according to amount. It is in this intermediate region that there is the range of therapeutic influence conveyed by the vehicles we call drugs. We think of these as forms of matter; we forget that matter is, to a large extent, merely a means of conveying energy. That which we term chemical constitution is the way in which energy is held, ready for release. It is by the relation of the latent energy of the agent to the energy being released in the tissue, and to the effect thus produced upon function, that one wide scope for the influence of drugs can be readily perceived. I think the facts we can discern justify the statement that treatment by drugs would be correctly named "dynamical therapeutics."

Although what we call "matter" is chiefly effective because it is a vehicle for energy, we must remember that certain elements are essential for its conveyance, and essential also

for the maintenance of the nutrition of the tissues. In the functional action of all structures in the body, molecules are constantly passing off, which, having formed with oxygen lower compounds and having yielded their latent energy, are useless, and these are constantly being replaced by others, formed under the influence of life from the material which the plasma has brought to the vicinity of the tissue-elements. The constituents of some "poisons" may enter into the molecules of the tissues and thus disturb function by deranging the release of energy, and also derange structure. When arsenic is brought into contact with the nerve elements, whatever be its combination, it induces, first functional, and then structural changes in these, leading ultimately to their degeneration. We can only explain this by supposing that atoms of arsenic are taken up by the nerve substance in the course of the molecular renewal which attends functional action, "metabolism," it is called, and it is reasonable to suppose that this is due to its close relation to phosphorus, which we know is a constituent of the nerve material. It is possible that such substitution may take place in a slight degree without any injurious consequences, and even, in morbid states, with benefit. The influence on the nutritional process may be actually beneficial when this is already in some way deranged. It may perhaps promote the subsequent due assimilation of phosphorus, and make this more adequate and effective. Of course this is only a speculation. It is often useful to speculate when it is impossible to see.

These remarks have reference of course only to the medicinal agents which act through the blood. There is a considerable scope for thought regarding the action of agents which do not pass into the blood, which act only upon the surface, or on that portion of our real exterior which is within us, as the alimentary canal. In this region, moreover, there is some room for bringing conception to the test of

experiment. Many purgatives, of course, act through the blood, but chemical processes seem to take less part in the operation of saline aperients. These act rather by "flushing out," the liquid in which the salt is dissolved being merely added to by osmosis. The subject, indeed, requires more special study than it has received. The great service of salines seems to me to be effected by removing that which would be in part reabsorbed. They carry away bile, much of which would pass back into the blood. This is doubtless true also of the purgatives. Some years ago it was said that the old idea that mercury relieves the liver was a mistake, because experiment showed that it did not increase the secretion of the bile. Yet the old idea was based upon a fact—that it does increase the amount of bile which leaves the body. This would pass back to the liver by the portal vein; by its removal the liver will be as effectually relieved as if its secretion were increased, perhaps indeed the relief will be even greater.

One other phase of modern therapeutical thought is the contrast which is often drawn between the empirical and the rational in the use of drugs, and the disparagement of the former in comparison with the latter. It is a comparison which deserves consideration. In part it is a result of the influence of the scientific training which now dominates medical education. Beneficial as this training is, its influence may acquire a momentum that carries the effect too far for the good of those whose life is to be devoted to the hard and often routine work of applying knowledge. It is all very well to esteem the rational, but this esteem should not be allowed to cause an aversion from that which is precious, and yet cannot be called "rational" in the common, narrow sense of the word. The antithesis of the empirical and the rational is itself an error. It is an instance

of a common tendency to put a negative conception into a positive word. Because the empirical is not the rational it is conceived as irrational—the only simple negative we have, but one that implies far more than mere absence of that which is excluded. The "irrational" should be merely the "not rational"; it should mean only that for which an explanation cannot be discerned. In effect, it carries us as far beyond neutrality as does "unreasonable." No one means by "unreasonable" a simple absence of the quality of reasonableness; he means that which is opposed to reasonable. Not only is the empirical not positively irrational; it is doubtful how we are justified in considering that a truly rational element is absent from the empirical. The term "rational therapeutics" is applied to treatment in which a drug is given with success in accordance with preconceived ideas or theory. The theory may turn out quite wrong, although the result is the same. What, then, becomes of the rationality? In empirical therapeutics a drug is given because it is found by experience that in the particular condition it does good. Often we cannot even guess why. But the fact remains, and surely to act upon observed experience is as truly a rational proceeding as is action upon a theory which may be correct or incorrect. After all, the medicinal treatment which can be based upon any definite theory is small. How few are the drugs we can use to advantage which were not discovered by pure experience. In not one drug in twenty of those of most certain service can the use be traced to anything except unguided experiment. Our knowledge of these drugs, derived from the past,—and often from the distant past,—must be assumed to be the result of experiments innumerable, perhaps continued through the long centuries in which the human race has lived under the need to counteract disease by every available means. The need for food must itself have led to a knowledge of the physiological action of most

herbs of the field, and the habits of animals under observation may often have had the force of example. It is probable that since man became able to observe and to reason, every common herb of the field and fruit of the tree has been at some time tested, and thus by slow degrees the knowledge of a physical good and evil has been acquired.

It is strange, indeed, to note how far back goes the use of the drugs on which we most rely. Most of them can be traced far back into the distant past until they are lost in the blue mists which shroud alike the hills of Greece and the deserts of Arabia, or to the time when the world learnt its wisdom from the land where now the symbols of man's thought lie deep beneath the desert sand or stand silent in the cold moonlight of a long dead past. We smile at the popular herbal remedies. But it is to these that we owe the majority of our most useful drugs. I cannot conceive a therapeutist surveying a list of the chief drugs on which we depend in our daily work—and do not depend in vain—without a sense of wonder and perhaps of humiliation. We disinfect our rooms with burning sulphur; and so men did before the time of Homer. We purge sometimes with rhubarb, especially when some subsequent astringent influence is desirable, and so did the old Arabians for the same special reason. The value of castor oil in its chief use was familiar, probably for ages, to the natives of the East and of the West Indies before it was made known in Europe by a physician from Antigua one hundred and fifty years ago. Aloes was employed in the same way long before the time of Dioscorides and Pliny. The knowledge of the influence of ergot in parturition we owe to the peasants of Germany, and the use of male-fern for tapeworm goes back to the old Greeks and Romans. The employment of mercury in syphilis by inunction and fumigation, which our nineteenth-century therapeutists regard with such satisfaction, seems to go back

to the time of the Crusades, and it is said that its use can be traced in Malabar as far back as the ninth century. Podophyllum as a purgative we owe to the North American Indians. If we go through all the drugs on which we most rely we find the same story. Even in the case of those which are the latest additions to our resources, we find that, with very few exceptions, their use arose from what we must regard as pure empiricism. It was by accident that the local anæsthetic influence of cocaine was discovered. The unexpected results of simple experiment afforded us the chief use of antipyrin; and that which is perhaps the greatest practical discovery of modern times in the influence of drugs on disease—the use of bromides in epilepsy—was the result of a chance observation of its use on an allied state—also empirical. Precisely the same statement is true regarding the employment of iodide in syphilis. It arose from an almost random trial of the influence of burnt sponge on goitre. To this day we are without any rational perception of their mode of action. I yield to no one in my sense of the importance of the rational in therapeutics; but we need to be careful lest, in contrasting the rational and the empirical, we allow our esteem for the one to induce a depreciation of the other. We can afford to despise no source or kind of help, nor to permit our estimate to be prejudiced by the many warping influences to which our thought is liable.

Any attempt to frame a definition of rational therapeutics will, I think, have one effect. It can hardly fail to raise a doubt as to the propriety of considering that a theory to explain an empirical discovery makes the therapeutics rational. It is very easy to frame a theory of the action of a drug, and it is easy to extend this theory to the nature of the disease in which the drug does good, and at the same time to ignore the many other possible ways in which the effect may be produced, and so to build from an uncertain

foundation an edifice altogether unstable. When I recommend a drug I am often asked "How does it act?" Occasionally I can give some adequate reason, but I am generally compelled to answer, "I do not know; it is often useful in this condition." Sometimes I can add, "There are several ways in which it may act." Sometimes I am obliged to say, "I have no idea how it does good." It has not been my privilege to add much to our therapeutical resources, but the few agents I have recommended have been based on pure empiricism. Many observers have confirmed the statement which I made more than twenty years ago regarding the occasional service of borax in epilepsy. In inveterate cases which do not yield to bromide, borax sometimes does good that is definite and distinctly greater than that which bromide produces in those patients. But I cannot say why. It was one of the many things I tried, simply as a peasant might try in succession a number of herbs. Further, the diminution in the tendency to the distressing pains in locomotor ataxy which is caused by the regular administration of chloride of aluminium is so distinct that I have little doubt that the time will come when this drug will find a place in the Pharmacopœia. But I had no other reason for trying it than the fact that some analogue suggested it. Of the rational we have here no trace, although I should take objection to the difference involved in the application of the epithet "irrational."

There is much more I should like to say for which time fails. I must content myself with expressing the hope that you will endeavour to apply what I have suggested to you. I feel sure that such a Society cannot do more for its members, and cannot in any way do more for the profession outside, than by carefully considering what drugs are found, by the personal experience of each one, to be of real use, by combining the results of personal observation

and by endeavouring in the future to organise mutual help in respect to the new therapeutic agents which are bestowed upon us by the earnestness and energy of others.

I count it a high privilege to teach, but a privilege far higher to help men to teach themselves. It is easy to lose that which is merely received. That which is acquired is persistent. Knowledge personally gained becomes part of a man's mental self—not only as knowledge, but as power. It rises in its influence into every branch of the complex activity of practical life, and it becomes effective through its indirect effect even where we cannot trace its presence.

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